VOLUME 65 • MARCH 1957 • NUMBER 3

A.M.A. ARCHIVES OF OTOLARYNGOLOGY

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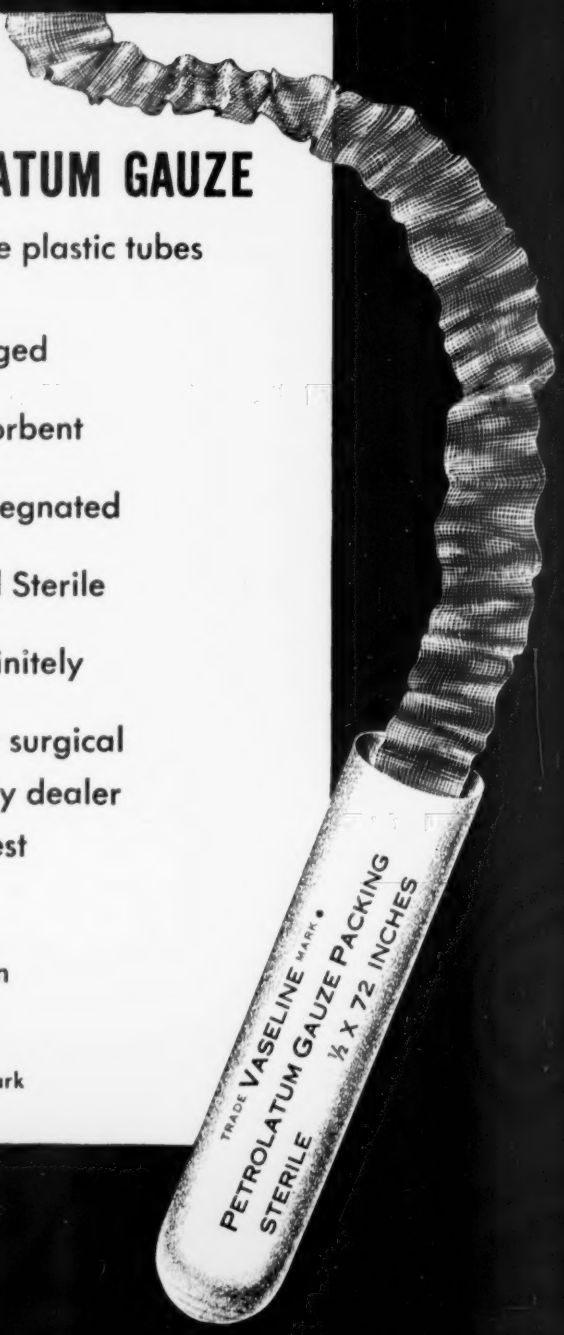

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Checks, money orders, and drafts should be made payable to the American Medical Association, 535 North Dearborn Street, Chicago 10.

AMERICAN MEDICAL ASSOCIATION Publication

Published monthly by the AMERICAN MEDICAL ASSOCIATION. Editorial and Circulation Offices: 535 North Dearborn Street, Chicago 10, Illinois. Publication Office: Thompson Lane, Box 539, Nashville 1, Tennessee. Change of Address: Notice to the circulation office should state whether or not change is permanent and should include the old address. Six weeks' notice is required to effect a change of address. Second-class mail privileges authorized at Nashville, Tenn., Aug. 6, 1956.

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The Uses and Abuses of the Adrenocortical Steroids in Otolaryngology

THOMAS F. FLYNN Jr., M.D., Woodbury, N. J.

The glucocorticoids recently have attained great acclaim in the armamentarium of the otolaryngologist. There is no doubt that they have their place as therapeutic agents in certain diseases with which we are concerned; however, the misuse of these hormones is widespread. I say this because these hormones are being utilized in the treatment of diseases in which the rational of their use is not justified. To understand the effect of the corticosteroids some basic concepts must be understood (Fig. 1).

As their name implies, these hormones emanate from the cortex of the adrenal gland, the latter being composed of several layers. It is believed that desoxycorticosterone (Doca) is produced in the granulosa layer and cortisone and hydrocortisone (Compound E and Compound F, respectively) in the zona fasciculata. Sex hormones, such as estrogens and androgens, emanate from the reticular layer and epinephrine, naturally, from the medulla. Of the 28 or more steroids known to date, 6 possess appreciable biological activity; they are cortisone, hydrocortisone, corticosterone (Compound B), dehydrocorticosterone (Compound A), desoxycorticosterone, and 11-deoxy-17-hydroxycorticosterone (Compound S). These steroids are similar in basic structure, with differences in their physiological activity being due to slight variations in the side-groups attached to the basic steroid nucleus. The two main end-products of corticosteroidgenesis used mainly today are cortisone and hydrocortisone. Their chemical structure resembles that of cholesterol in that both are steroids. It is obvious that they differ in only a single detail (Fig. 2). Cortisone has a ketone group, while hydrocortisone has a hydroxyl radical at position 11 of the steroid configuration. It is interesting to note many of the newer derivatives are identical except for a minor change at one of the positions on the benzene ring (Fig. 3).

To appreciate their use in disease, it is important to be mindful of their action. The present concept, as outlined in the schematic drawing (Fig. 4), is that the secretion of cortical hormones is mainly mediated through the stimulation of the adrenal cortex by the adrenocorticotropic hormone from the anterior pituitary. The anterior pituitary in turn is controlled to some degree by the hypothalamus, which again in turn is controlled by the higher cerebral centers. Epinephrine also stimulates the anterior pituitary via sympa-

Accepted for publication July 24, 1956.

Read before the Section on Otolaryngology at the 190th Annual Meeting of the Medical Society of New Jersey in Atlantic City, N. J., on May 16, 1956. This paper has been released for publication here through the courtesy of the *Journal of the Medical Society of New Jersey*.

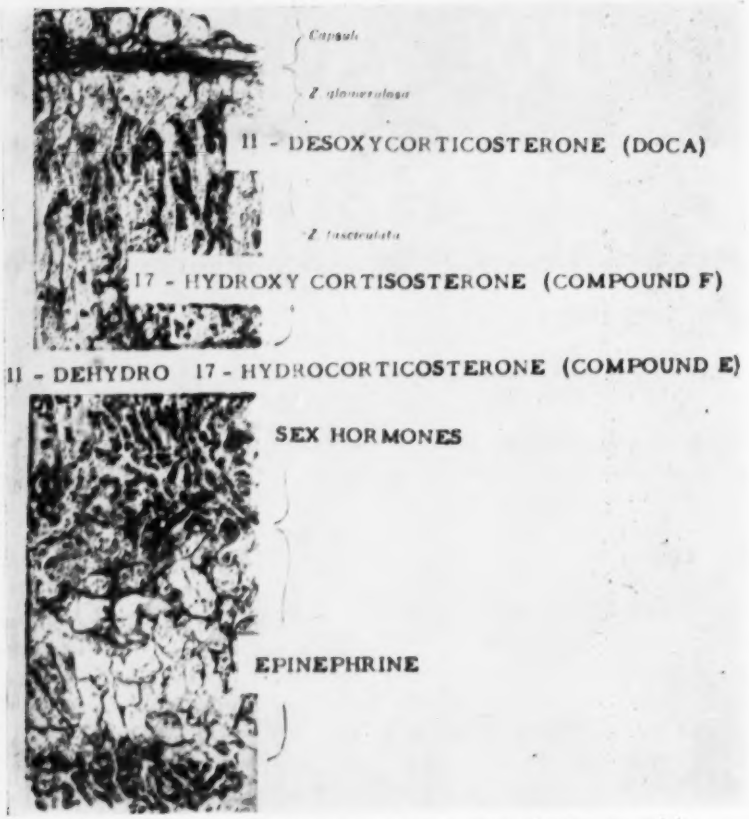
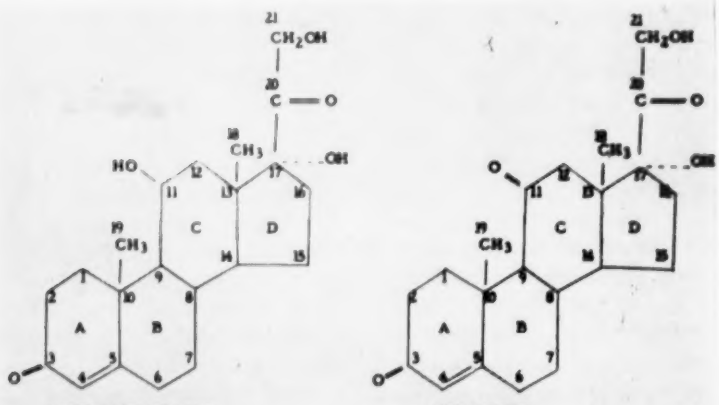


Fig. 1.—Microscopic section of the adrenal gland showing the origin of the steroids.



It is obvious that they differ in only a single detail. Compound E has a ketone (C : O) group, while compound F has a hydroxyl (OH) radicle at position 11 of the steroid configuration.

Figure 2

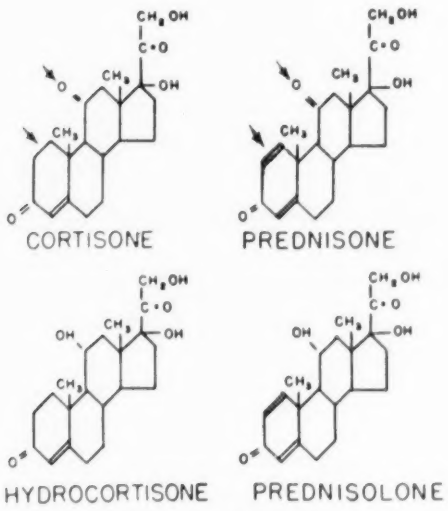


Fig. 3.—Similarity of the steroids, with the only change in the benzene ring.

thetic release in response to stress. Selye has suggested also that the somatotropic hormone from the anterior pituitary plays a role in releasing cortical steroids.

Naturally, it would be incomplete to omit a discussion of Selye's theories, in view of the fact that they explain the exact action of these hormones. Since his works have been read and reread, it should suffice to say that the body reacts to all stress in a stereotypical manner. This reaction is a "Call to Arms" of the body's defense forces, and he named this the Alarm Reaction. The Alarm Reaction is the first stage of a more prolonged general adaptation syndrome. The latter comprises three distinct stages: Stage 1, the Alarm Reaction itself, in which adaptation has not yet been acquired; Stage 2, the stage of resistance, in which adaptation is optimal, and, finally, Stage 3, the stage of exhaustion, in which the acquired adaptation is lost again. This last stage, however, if reached terminates in destruction, be it local tissue (as demonstrated in the local adaptation syndrome) or the body itself (as in the general adaptation syndrome). During the Alarm Reaction the corticosteroids are secreted, and Selye classifies them as glucocorticoids and mineralocorticoids. The glucocorticoids, such as cortisone and hydrocortisone, are antiphlogistic, and the mineralocorticoids, such as desoxycorticosterone, are prophlogistic. The mechanism of this alarm reaction is demonstrated in Figure 5. The stressor, which may be physical, chemical, or biological, acts upon the target, which is the body or some part of it. Through some unknown pathway, labeled by the question mark, the first mediator travels from the directly injured target area to the anterior pituitary. It notifies the latter that a condition of stress exists and thus induces it to discharge corticotropin (ACTH). It is quite possible that this "first mediator" of hormonal defense is not always the same. In some instances it may be an epinephrine discharge; in others, a liberation of histamine-like toxic tissue metabolites, a nervous impulse, or even a sudden deficiency in some vitally important body constituent, such as glucose or an enzyme. Corticotropin stimulates the adrenal cortex to discharge corticoids. Some of the prophlogistic corticoids stimulate the proliferative ability and reactivity of connective tissue; they enhance the inflammatory potential. In this way they put up a strong barricade of connective tissue so that the body is protected against further invasion of the stressor agent. The antiphlogistic hormones, such as cortisone and hydrocortisone, are secreted in a greater percentage, and they are antagonistic to the prophlogistic corticoids. These inhibit the ability of the body to place granulomatous barricades in the path of the invader; in fact, they tend to cause involution of connective tissue with a pronounced depression of the inflammatory potential, and thus they open the way to the spread of infection. This is the reason that when cortisone and hydrocortisone are employed in the treatment

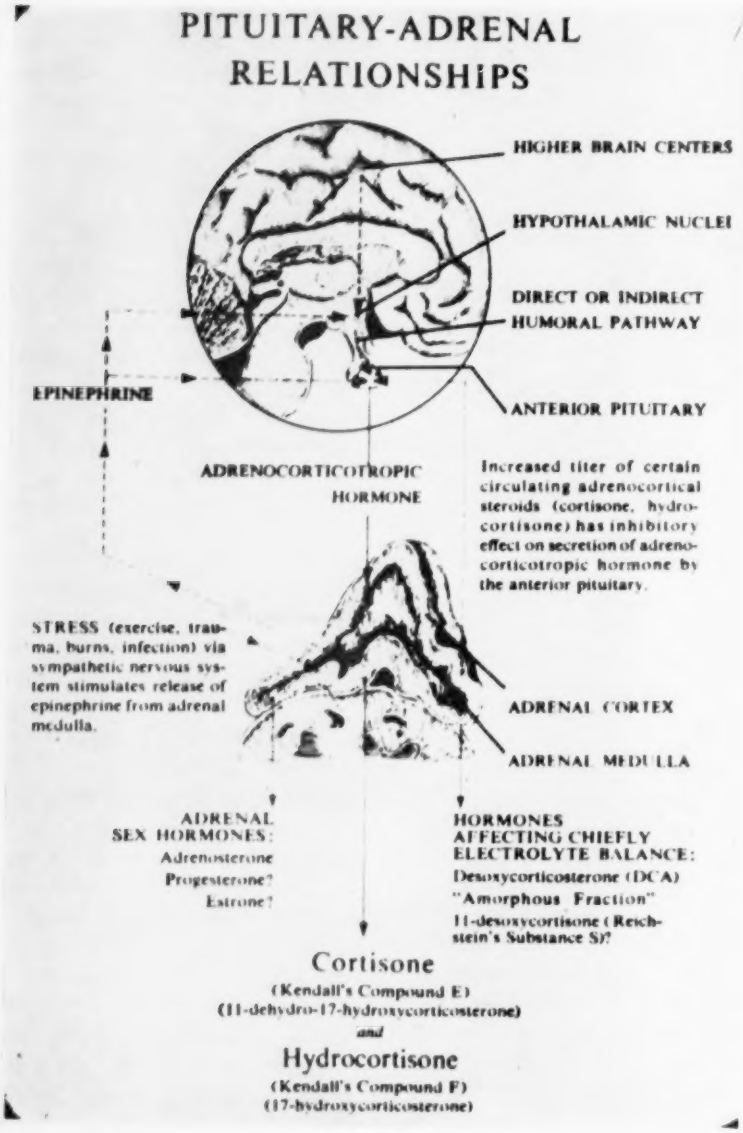


Figure 4

of infections they should be well covered by antibiotics.

Certainly the administration of the corticosteroids in the presence of a coexisting contraindication is an outright abuse. The most prominent contraindication is the existence of a peptic ulcer because of the possibility of perforation, since the glucocorticoids, cortisone, and hydrocortisone, as mentioned before, tend to cause involution of the connective tissue. Heart failure, hypertension, and renal insufficiency are contraindications mainly because of the water retention frequently seen. Recently we treated a patient with prednisone, the anlage of cortisone,

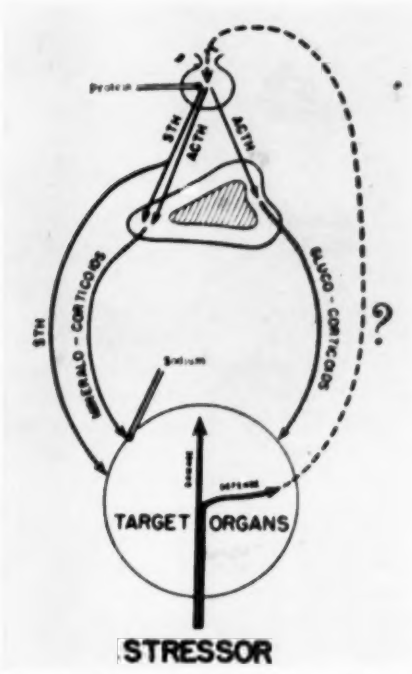


Fig. 5.—Selye's Alarm Reaction: Schematic diagram illustrating the principal interrelations between the hypophysis, the adrenal cortex, and the peripheral target organs during the general adaptation syndrome. (Slightly modified after H. Selye, "First Annual Report on Stress," Acta, Inc., Medical Publishers, Montreal, Canada, 1951.)

who began to develop personality changes, which subsided after the drug was discontinued. Cortisone has precipitated psychoses and therefore should not be used in any case with a preexisting psychotic tendency. Varicella is a newer addition to the list. Recently three deaths have been reported in children who developed chicken pox while under cortisone therapy for some other condition. Autopsies revealed chicken pox lesions in every organ of the body. This spread of infection is manifested also in active tuberculosis. Severe diabetes is a contraindication and may even be precipitated by the use of these hormones. Glycogen storage is prominent and hence the need for more insulin. In normal persons this need is met by a normally functioning pancreas.

The toxic or warning signs of overdosage on prolonged administration are listed as follows:

1. Cushing-type response
   (*a*) Centripetal fat distribution
   (*b*) Moon face
   (*c*) Sunfish mouth
2. Acne; hirsutism
3. Hypertension
4. Personality changes
5. Fluid retention; increased weight
6. Glycosuria

Hence prior to the institution of therapy, especially higher doses, a competent history must be taken with the contraindications in mind. The patient has to be watched for weight gain, edema, and hypertension. Some authors have suggested that prior to therapy blood sugar determinations and chest x-rays be performed. If higher dosages are used the patient is usually taken to the hospital, where blood pressure, intake and output charting, daily weight, and urine can be watched for signs of excessive hormonal administration. Administration during pregnancy should be guarded because of the edema factor. The diet should consist of a low-carbohydrate and high-protein content. Sodium retention is common; therefore restriction of salt and limitation of fluids are beneficial. One of the greatest misuses in the administration is the abrupt cessation of the therapy. This produces evident adrenal depression because the cortex is being suppressed during administration. The withdrawal should be gradual in steplike fashion so that this abrupt depression does not occur.

With prolonged use the adrenal cortex may be suppressed to a great degree; however, recent studies show that cortisone-induced atrophy is reversible. This reversibility may be inversely proportionate to the length of therapy. Many men advocate intermittent use of corticotropin during cortisone therapy to stimulate the adrenal cortex. This is not needed in short-term use such as a week or 10 days. Figure 6 depicts the suppressive effect of cortisone and the stimulating effect of corticotropin

Fig. 6.—Action of cortisone and corticotropin on the adrenal cortex.

on the adrenal cortex. It is important to remember that this suppression exists and with surgical procedure more suppression is likely and, consequently, if the patient is receiving cortisone therapy it has to be increased prior to surgery.

Because of the depression of the inflammatory potential mentioned before, I think the administration of these hormones in ordinary infections constitutes an abuse. Unless the patient is in a terminal stage or the disease is a rapid fulminating type, such as meningococcemia or meningitis, it is well not to use the corticosteroids. If they are used early in infection, they must be well fortified with antibiotics and there must be a specifically known antibiotic which is effective against the organism causing the infection. Today successes are being obtained by the use of low doses of the corticosteroids, especially the newer anlages, such as prednisone and prednisolone. High doses preclude side-effects, and therefore high doses constitute an abuse of these drugs except where the disease warrants higher doses and the patient is hospitalized. We must remember that these hormones in the majority of instances offer only temporary relief and consequently cure nothing. In many chronic infections, such as bronchiectasis and many forms of sinusitis, alleviation of symptoms may be obtained but spread of infection may also be obtained, which, no doubt, is undesirable and harmful. Chronic dermatological conditions about the face and especially the ear, such as verruca vulgaris, keratosis follicularis, alopecia areata, and acne vulgaris, elicit no response therapeutically with the use of the corticosteroids. Allergic skin responses, on the other hand, show striking therapeutic effects, but if the offending allergen is not removed, recurrences will undoubtedly persist. True chronic vasomotor rhinitis of a nonallergic origin and hyperplastic sinusitis have to date showed no response to these hormones. One of the commonest misuses is the administration in allergic responses as a substitute for routine antiallergic therapy. Nothing is gained, and certainly nothing is cured. After cessation signs and symptoms will reappear. The glucocorticoids can be used in conjunction with desensitization and antihistamines and on occasion are beneficial. Their routine use is to be frowned upon; however, during an acute attack of a severe allergic response these hormones can and will tide the patient over until desensitization measures can be instituted, but it must be remembered that the usual case of allergic rhinitis or sinusitis can be controlled adequately by the proper antiallergic therapy, and hence the corticoids are not needed.

Certainly there is no place for the glucocorticoids in the treatment of deafness, even though they have been tried in cases of otosclerosis and other types. They also have been used in the treatment of tumors. The tumors with which we are usually concerned are the lymphomas. Lymphosarcoma responds to cortisone therapy by the rapid recession of enlarged lymph glands, but after cessation of therapy—and usually before—the nodes return to their former size. The value here is only of a temporary nature. If a rapid reduction in the size of the nodes is desired or response to x-ray is no longer noted, cortisone can be tried for palliation.

In otolaryngology, the most gratifying results are produced by the use of the corticosteroids locally; however, systemic administration has proved to be of value

in many entities. The bronchial tree manifests some amenable disorders. All of us see many asthmatics, either for bronchoscopic examination or for local treatment of an associated sinus infection, and have been deluged with literature, pro and con, regarding the corticoids. Since the management is usually assumed by the internist, it should suffice to say impressing results are being attained, especially with the newer derivatives, such as prednisone and prednisolone. Even over extended periods of treatment, maintenance has been obtained without obvious difficulty; nevertheless, it must not be forgotten that other antiasthmatic measures must be enforced. The intravenous route for status asthmaticus has been found to be lifesaving. Such conditions as pulmonary fibrosis and intractable emphysema without clinical signs of bronchospasm respond poorly to the corticosteroids. Loeffler's syndrome, since it is an allergic manifestation, has showed some response; however, not enough literature is available for an accurate evaluation. Bronchography with the use of the iodine products, such as chloriodized oil (Iodochlorol) and iodized oil (Lipiodol), is sometimes associated with sensitization or iodism. Park and his co-workers[13] used corticotropin prior to bronchography in known iodine-sensitive patients and found that it decreased the sensitivity so that the procedure could be carried out in an orderly fashion.

The routine use of steroids in ordinary infection is certainly to be avoided. There have been several theories on the mechanism of the hormones in infection; but no matter what the mechanism may be, the fact remains that the inflammatory potential is markedly depressed and consequently the spread of the infection is imminent. In severe fulminating disease, such as meningococcic infections for which specific antibiotics are available and in a case where the patient's condition is so severe prognosis is doubtful, the steroids can be lifesaving. In the evaluation of cervical adenopathy, we encounter many cases of mumps. Mumps orchitis is a common complication and is another condition in which the steroids play a part, even though an effective antibiotic against the viral agent is lacking. Again it must be stressed that the treatment should be used for the shortest possible time because the patient may demonstrate striking clinical improvement while the organism causing the infection flourishes. The Waterhouse-Friderichsen syndrome, a serious complication of meningitis, needs heroic therapeutic measures with blood transfusions and intravenous hydrocortisone. This is not so far from the field of otolaryngology when one is dealing with mastoid disease and its complications. I treated a case such as this at the Graduate Hospital prior to the advent of hydrocortisone but was unsuccessful. Caution must be applied when utilizing the corticosteroids in infection, for a mild infection may develop into a lethal disease.

Local applications in infections prove much more promising. Many antibiotic steroid combinations are available today in the form of nasal sprays, ointments, and solutions. Some of the best results are noted in the ear and the nose. Review of the recent literature points out that the best results are obtained by the use of a 2.5% concentration of hydrocortisone. Local cortisone has given precedence to the newer anlages, such as prednisone and prednisolone. The latter, the anlage of hydrocortisone, is most effective in a lower concentration of 1%. These ointments when applied locally to such conditions as eczema and dermatitis of the external canal are beneficial; however, lichen planus, verruca vulgaris, and herpes simplex have showed little or no response. Senturia[6] found that external otitis responded well to the antibiotic preparation containing hydrocortisone acetate and neomycin sulfate (Neo-Cortef), but he encountered some resistant cases. The resistant cases were usually of a fungus type and did not clear up until typical antifungicidal drugs were

used. I have used both Neo-Cortef and hydrocortisone (Cortil) ointment and lotion and have been well impressed with the results. Fluorohydrocortisone used locally is most effective in the concentration of 0.5%, but I have been disappointed in its effectiveness and have seen many more reactions with this than with the other corticoid preparations. In the less severe types of external otitis, local hydrocortisone preparations can be used in the 1% concentration, and if combined with antibiotics, such as Neo-Cortef, Terra-Cortril (a compound containing 3% oxytetracycline hydrochloride and 1% hydrocortisone), and the like, a 0.5% concentration is sufficient. With severe otitis, on the other hand, the 2.5% concentration gives the best results. In fulminating cases, systemic hydrocortisone or prednisolone is of value. It must be remembered that this type of regimen provides only temporary relief and certainly does not replace some of the older methods for the treatment of external otitis.

Postoperative fenestration and mastoid cavities with secondary infection when exposed to the corticoids dry more readily. Granulation tissue is reduced to a minimum except, of course, if the infection is due to fungus. Success in the treatment of chronic otitis media has been reported. The reduction of the granulation tissue allows easy access to the middle ear, where curettage of disease tissue is facilitated. This is well and good; nevertheless, there are few chronic draining ears that do not have mastoid pathology as their precursor and hence will respond to nothing less than a modified or radical mastoidectomy.

Systemic corticoids have been tried in several disorders of the ear and have been found useless. Rosen and Feldman tried, as they report in an article written in 1953,[7] to determine the effects of cortisone on hearing. The hearing threshold was unchanged, but the threshold of discomfort was elevated. I can see no significance to this limited study. In 1952, Riskaer[5] reported 12 cases of otosclerosis treated with corticotropin to no avail. I can see no indication for corticoid therapy in deafness except possibly where the latter is on an allergic basis.

Allergic reactions, notably, subside quickly with a corticoid regimen. Drug eruptions, anaphylactic shock, hay fever, and the like, respond well in the acute stages. In the case of anaphylactic shock from penicillin injection or other drugs, 10 mg. of corticotropin can be given intravenously along with the usual method of treatment, i. e., the administration of epinephrine, oxygen, and aminophylline. Injectable hydrocortisone prepared in the form of hydrocortisone sodium succinate (Solu-Cortef), 100 mg., can be used intravenously. It has been reported as lifesaving in acute drug reactions. This is an excellent hormone to add to the emergency tray. The corticosteroids do not cure allergic conditions, but they do afford symptomatic relief as a preliminary procedure prior to or in conjunction with standard antiallergic therapy. This holds well in the rationale of treatment of the nasal allergies and hay fever. Marked improvement of symptoms results quickly after a regimen of 60-80 mg. of hydrocortisone daily or 20-30 mg. of prednisolone, but, as I have said before, this is only temporary and after withdrawal of the hormone the symptoms will recur. Sometimes during an acute attack therapy such as this is needed in persons who do not react well to the usual desensitization methods.

Hydrocortisone is known to shrink nasal polypi both by local and systemic use. Silcox,[10] in an evaluation of local hydrocortisone preparation in the nose, reported extremely effective symptomatic relief in allergic rhinitis with the use of Vasocort, a preparation containing two vasoconstrictors and 20 mg. hydrocortisone alcohol per 100 cc. We exhibited these results at the American Medical Association Convention held in San Francisco in June, 1954, and since then I have had even greater success.

Other preparations with varying concentrations of hydrocortisone have been used; however, reactions have been noted. I have had very little occasion to use the submucosal injection of hydrocortisone. Seminov injects 2.5 to 5 mg. of hydrocortisone into the anterior end of the inferior turbinate and states he gets very good results. Wall and Shure [11] noted a favorable response in 80% of 65 cases treated by injecting anywhere from 2.5 to 25 mg. of cortisone suspension. The injection of the larger doses produces unwarranted systemic reactions. I do not believe this type of procedure is needed in view of the fact that satisfactory shrinkage of polypi can be afforded by the local intranasal application or the systemic use of the newer less toxic derivatives. Nevertheless, one must not forget that corticoid therapy, either local or systemic, does not replace polypectomy, in the majority of instances, and that still in many polypectomy is the treatment of choice.

On the other hand, many patients with nasal polypi and severe allergic rhinitis are not amenable to surgical or allergic measures and can be maintained on 10-15 mg. of prednisolone daily for many months without harmful effects. This type of therapy has been useful in many for more than a year without adverse effects, although higher doses are usually needed to control symptoms and hence would not justify long-term use.

Intranasal use of hydrocortisone in combination with antibiotics has been impressive. Preparations such as Trisicort Biomydrin F and the like have in my hands been a great advance in the management of purulent rhinitis. I do not believe these are indicated in the usual "stuffy nose" or rhinitis associated with the common cold. The less nose drops or intranasal medication is used the better off the patient will be, especially in a mild upper respiratory infection. The systemic use of vasoconstrictors has been successful in alleviating nasal congestion and is certainly less irritating.

With many of these nasal disorders mentioned there is on occasion an associated sinus involvement, and a word has to be said concerning the value of the corticosteroids in sinus disease. Their place, limited though it may be, does not supplant other measures, such as conservative surgical procedures which promote ventilation and drainage. The best results have been obtained in allergic sinusitis, and the regimen here is the same as that for allergic rhinitis. Short-term results can be obtained with the use of 20 mg. of hydrocortisone, three times daily, or 10 mg. of prednisolone, 3 times daily, and naturally it is best used during the period of acute reaction. Intrasinal instillation of a 2.5% hydrocortisone acetate suspension has been used with little value. The corticoids will reduce the size of the antral polypi, but with the cessation of the drug the polypi will recur unless the proper institution of antiallergic therapy has been begun. Even with this many will not recede. Certainly in chronic hyperplastic sinusitis the corticosteroids should be avoided because even though in some patients favorable results will be noted, these will be short-lived. The maintenance dose is usually so high that signs of toxication ensue.

The part of the digestive tract with which we are mainly concerned, namely, the mouth, pharynx, and esophagus, manifests some lesions which are amenable to corticoid therapy. The local use of hydrocortisone, especially in the mouth, is of little value. Many reports claim inconsistent results. I have found such lesions as aphthous ulcers to recede under systemic prednisolone therapy when other measures had failed. Fisher [12] recently reported the use of hydrocortisone locally in several conditions from glossitis to lichen planus, but actually aside from some "fair" results in "inactive" leucoplakia there seem to be little or no results from the majority of lesions treated. I have used local hydrocortisone ointment on the lichen planus lesion but have been grossly disappointed. Local lesions from ir-

ritation of dental plates, tooth paste, mouth wash, and the like, recover swiftly after the removal of the irritation; hence corticoid therapy is not justified.

Pemphigus rarely is seen in the mouth but does occur. Many have been reporting good results with the systemic use of prednisolone, but I have not had the chance to evaluate its efficacy in this disease.

Oral burns from acid or lye respond exceptionally well. Recently I treated a child who swallowed household ammonia and sustained marked pharyngeal edema, edema of the soft palate and buccal mucosa. After a regimen of hydrocortisone intramuscularly (10 mg. four times daily) the edema was not noticeable within 48 hours. Usually these go on into secondary infection if they persist. This same child was treated symptomatically for esophageal burns, and after five days of therapy a swallowing function was performed and was found to be normal. There was no esophageal erosion whatever. At the Underwood Hospital, Dr. R. Moore and I have treated several children in this fashion and have had remarkable results. Not enough information has been obtained to date for an accurate scientific evaluation. The patient should be watched closely while under this type of treatment, because Rosenberg, Kunderman, Vroman, and Moolten [3] reported a high incidence of perforation of the ulcerated esophagus in rabbits under cortisone therapy. Investigations so far today reveal that the incidence of strictures is lessened and the length of the disease is shortened; nevertheless, there is no doubt that more study is needed, so caution should be taken when utilizing these hormones for lye burns of the esophagus.

Corrosive burns also affect the larynx, in many cases producing laryngeal edema and consequent stridor. Many tracheotomies have been averted by the intelligent administration of the corticosteroids when dealing with laryngeal edema. Edema subsequent to prolonged bronchoscopy, allergic reactions, burns, and trauma from foreign bodies and such subsides readily with corticoid therapy; however, in stridor associated with acute infection the use can be harmful. Corticosteroids can change a mild laryngitis into a fulminating laryngotracheobronchitis, and subsequent overwhelming infection and death.

Edema of the facial nerve is another condition in which the corticoids have been utilized. I have treated two early cases of Bell's palsy with cortisone, and the paralyses subsided within one week. There is no doubt, in the majority of instances, that a self-limited condition usually recovers in a matter of weeks or months. Robison and Moss [2] treated two more cases with cortisone, with gratifying results, within one week. Rothendler [1] states that cortisone relieves the acute edema and inflammatory reaction of the facial nerve; hence in postoperative mastoidectomy and fenestration cases with facial damage the corticoids should be effective. No studies to this date are available on postoperative facial paralyses.

In otolaryngology, we are not too much concerned with arthritis, except when it involves the temporomandibular joint. Control of pain can be obtained by the injection of 15-20 mg. of hydrocortisone into the temporomandibular joint space. Care must be taken to avoid the superficial temporal artery. An x-ray of this area is helpful not only for diagnosis but also for studying the anatomic relationship prior to injection. A No. 22-24 gauge needle, 1½ to 2 in., is used with a rubber stopper attached to the needle ½ in. from its point to gauge the depth. With the patient's mouth open, the needle is inserted perpendicular to the mandible ½ in. anterior to the tragus and approximately ½ in. in depth. Pirkey [4] from Pittsburgh reported injections of 350 patients in this manner for pain associated with Costen's syndrome. He found that 316 patients obtained complete relief of pain within 24-36 hours. This lasted on an average of from five to six months. Needless to say that if the underlying cause is not corrected, such

as abnormal bite, the pain will recur. He based this rationale of therapy on the work of Bauer,[3] who showed that noninfectional, functional trauma to the temporomandibular joint promotes the development of osteoarthritis. In these cases where long-standing arthritis is present local injections of hydrocortisone are beneficial.

In conclusion, I should like to say that the investigation of the efficacy of the corticosteroids is certainly incomplete in the field of otolaryngology. This should inspire us to study the effects of this drug on various conditions encountered in our practice; however, this does not give us a license to use these hormones indiscriminately. If perhaps any study is undertaken, it should be performed with the proper control, for a study without a control is useless! The use of these hormones for conditions other than those which have shown clinical response is definitely abusing these hormones and endangering the life of the patient.

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Amyloid Tumors of the Larynx and Trachea

Review of Nine Cases with Detailed Report of One

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Primary amyloid deposits occur in many parts of the body, including the skin, tongue, heart, pharynx, urethra, bladder, and respiratory tract. Laryngotracheal sites are the commonest, and primary amyloid tumors of the larynx and trachea are not so rare as they were once believed to be. In the past several years a sufficient and increasing number of reports have appeared in the literature to attest to the fact. However, there does not seem to be any unanimity of opinion regarding the etiology, pathology, and treatment of this lesion, which presents definite diagnostic and therapeutic problems.

Stark and New[13] reviewed the case records of all the patients given the diagnosis of amyloid degeneration of the larynx at the Mayo Clinic up to 1948. The diagnosis of amyloid degeneration was confirmed microscopically in 15 of these cases, which constitute one of the largest series published. We have reviewed the surgical files of the Mayo Clinic from 1949 through 1955 and found records of nine additional cases of amyloid tumors of the larynx and trachea. It is the purpose of this paper to review these additional cases and to report in particular one case in which a skin graft was used to reconstruct the laryngeal lining, as had been done by one of us (Figi[8]) in a previous instance.

Accepted for publication Oct. 4, 1956.

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Review of Pathology

Amyloid originally was given its name in 1853 by the pathologist Virchow, who noticed that this substance reacted similarly to starch when iodine and sulfuric acid were applied to the tissue in which it lay. Amyloid is homogenous and amorphous. It usually shows a special affinity for Congo red and stains metachromatically with methyl violet and methyl green.

The structural nature of amyloid has not been established definitely,[3] most probably because the material is difficult to isolate and because apparently its structure must vary with the pathologic state in which it occurs. The variable staining reactions of primary amyloid point to this latter fact. All amyloid is composed mainly of protein-like materials and amino acids, and a small amount of polysaccharides similar to chondroitic acid.

The cause of amyloid accumulation remains obscure.[6,8,10] The deposits occur mainly in smooth muscle and connective tissues of blood vessels and the surrounding tissues. Hypersensitivity, perverted protein metabolism, and abnormal mesenchymal or other tissues have been implicated in its pathogenesis.

The symptoms, when present, are due to physical factors—the size and location of the tumor.[12] Piecemeal removal with or without electrocoagulation,[5] roentgen therapy,[1] treatment with cortisone and corticotropin (ACTH),[9] and excision with skin grafting of the denuded area are some of the methods of treatment reported.[1]

Classification

There have been many classifications of amyloidosis.[3,4,7,9,11] It may be categorized simply as primary amyloidosis or secondary, and as amyloidosis associated with multiple myeloma or a solitary amyloid tumor. The classification of Dahlin,[2] which follows, is based thus on distribution; and its primary type would include most cases of laryngeal amyloidosis.

1. Primary amyloidosis
   a. Systemic
   b. Focal or tumefactive
2. Amyloidosis complicating multiple myeloma
   a. Systemic
   b. Focal
3. Secondary amyloidosis

New[8] classified amyloid tumors in the respiratory tract according to the locations of the deposits, and thought that they could occur either in association with amyloid degeneration elsewhere in the body or as isolated lesions. Such deposits may be divided into three groups: (1) the diffuse subepithelial infiltration of amyloid, (2) the tumor-forming amyloid deposit, and (3) amyloid degeneration in a preexisting tumor.

Present Series

A review of the surgical files of the Mayo Clinic for the years 1949 through 1955 revealed nine cases of amyloid tumors of the larynx and trachea which are additional to those found by Stark and New[13] up to 1948. In this small series the sex incidence was practically equal, there being five women and four men. The majority of the patients were between the ages of 50 and 70 years at the time the diagnosis of amyloid tumor was made. The youngest in this series was 24 years old, and the oldest was 71. The symptoms were dependent on the size, shape, and location of the tumor. Hoarseness was the chief complaint in eight of the nine cases. Dyspnea, so common as a symptom in reports of other cases of this disease, and almost invariably severe in advanced amyloid disease of the larynx, was absent from this series. The duration of symptoms was 4 days to 10 years, averaging about 4 years.

In eight of the nine cases, the lesions were intralaryngeal, located on the true or false cords. One was subglottic. The intralaryngeal lesions were treated under suspension laryngoscopy in most cases by excisional biopsy with or without electrocoagulation of the base. One was treated roentgenologically, but this method was ineffective in arresting the growth of the tumor. The method of treatment used for the subglottic tumor will be presented in detail to illustrate an application of the principles of reconstructive surgery to this problem.

Report of Case

A woman, aged 24 years, had been hoarse intermittently for several years and constantly for three months before being seen at the clinic. Two months before her admission to the clinic a growth diagnosed as a hemangioma had been removed from the anterior commissure under direct laryngoscopy. Tracheotomy was necessary after this procedure because of obstruction of the airway. The tracheotomy tube was removed in one week, but the patient remained practically aphonic and complained of dyspnea and discomfort in the upper part of the chest.

Examination with the aid of suspension laryngoscopy revealed a bilateral thickening in the subglottic region and webbing below the cords. The mucous membrane overlying the tumor mass was slightly nodular in appearance. Biopsy and tracheotomy were performed at this preliminary examination. The tissue removed was diagnosed microscopically as that of amyloid tumor.

Exploratory thyrotomy was performed and the amyloid tumor was removed from the subglottic region, mostly by sharp dissection but partly by curettage. A cigar-shaped stent was fashioned from sponge rubber. A moderately thin split-thickness skin graft was removed from a hair-free part on the abdomen and cemented to the rubber stent with Mastisol, a mastic varnish. This stent was then inserted to cover the denuded part of the subglottic region, and the larynx and soft tissues of the neck were closed tightly. Immobilization of the stent was secured by stainless steel wires

passed through the neck and tied tightly over the gauze dressing on the front of the neck. The stent was left undisturbed for nine days, during which time the patient was fed through a nasogastric tube.

On the 10th day, the rubber stent was removed through the mouth with use of suspension laryngoscopy after withdrawal of the through-and-through wire sutures in the neck. The graft had taken well and completely covered the denuded subglottic region. A similarly shaped piece of sponge rubber was fashioned and covered with a rubber condom. This was inserted through the mouth into the larynx as a dilator. It was anchored to the tracheal cannula by a heavy nonabsorbable surgical (silk) suture passed lengthwise through the sponge rubber.

The dilator was left in place for 10 days, and then was removed through the mouth. Because the skin graft tended to shrink excessively, the dilator was reinserted about two and one-half weeks later and left in place for an additional three weeks.

In follow-up correspondence one year later, the patient stated that the volume and quality of her voice were good. Her airway was adequate, and she was leading a normal family life. Her only complaint was the occasional coughing up of small crusts such as sometimes tend to form on laryngeal skin grafts.

Summary

Stark and New found records of 15 proved cases of amyloid tumors of the larynx and trachea in the surgical files of the Mayo Clinic up to 1948. We found records of nine additional cases in the files for 1949 through 1955. We have presented in detail one such case in which the tumor mass was excised through a thyrotomy and the denuded area in the respiratory tract was resurfaced with a split-thickness skin graft.

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Fenestration of the Oval Window for Increasing Sound Conduction to the Cochlea

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In a recent communication [1] observations were reported on the technique, indications, and results of successful fenestration of the oval window in 10 cases of otosclerotic deafness. In these 10 cases, fenestration of the oval window restored the hearing after all attempts to mobilize the stapedial footplate had failed. In five of the patients in this series the crura had fractured, while in the remaining five the crura were intact. The hearing improved in these patients from preoperative levels of 65 db. loss to 15 db. postoperatively, and the improvement has been maintained for periods up to a year.

Accepted for publication Oct. 11, 1956.

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The fenestra ovalis opening is made by penetrating through the center or the inferior margin of the stapedial footplate into the vestibule for less than a millimeter (Figs. 1 and 2). The previously cited report described the use of the sharp-pointed explorer in creating the fenestra of the oval window. Through the fenestra thus created, one sees into a dark space, the vestibule, from which perilymph can be seen to escape. When a small pledget of moist cotton is placed in the round-window niche and is pressed gently in and out toward the round-window membrane, perilymph can be seen moving back and forth in the oval-window fenestra.

The purpose of this paper is to describe another new instrument for fenestration of

Fig. 1.—Oval-window fenestra created through the body of the stapedial footplate.

Fig. 2.—Oval-window fenestra created through the periphery of the footplate.

Fig. 3.—To create the fenestra of the oval window via the stapedial footplate, both the explorer (left) and the fenestrator (right) can be used.

the oval window and to indicate unusual pathological conditions at the footplate which can also be successfully overcome by fenestration of the oval window, with restoration of hearing.

The new instrument, called the oval-window fenestrator (Fig. 3), is shaped like a crochet hook and is used in the same way and at the same sites as the pointed explorer. The fenestrator is held firmly, but not tightly, with the cutting edge of the hook in the long axis of the footplate. In making the fenestration, care must be taken not to penetrate the oval window for more than a millimeter.

As the hooked fenestrator is removed from the vestibule, it carries outward with it a small piece of the stapedial footplate. Sometimes the fenestra is enlarged by a second penetration of the instrument.

This delicate procedure must be practiced on the cadaver until complete control of the instrument is achieved.

Occasionally, when the footplate is very bony and cannot be penetrated by the fenestrator, the explorer can be used first to pierce the bone sufficiently to make an easier pathway for successful penetration of the fenestrator.

The second reason for this communication is to indicate oval-window fenestration with restoration of hearing in two types of cases other than the usual bony otosclerotic stapedial obstruction. The first type of otosclerotic deafness is caused by fibrous, rather than bony, union of the footplate to the surrounding bone. Lindsay has reported[2] early cases of otosclerosis due to stapedial vestibular fibrosis, which accounts for the slight movement of the stapes during mobilization in some cases. In cases of fibrous fixation, the stapes seems to yield to increasing pressure on the neck, but this maneuver seems only to stretch the elastic fibrous tissue and the footplate then assumes its former fixed state, with no improvement in the hearing. This situation has been noted by Bellucci[3] and others during operation.

One such case is the following, already reported in a previous communication.[1]

On July 19, 1955, at operation the right stapes was found to be rigid. Increasing pressure on the stapedial neck caused the tendon to move freely, but the footplate assumed its former fixed state, with no improvement in hearing. Greater pressure on the anterior aspect of the neck finally caused fracture of both crura, and the hearing remained unimproved. The explorer was then applied to about the center of the body of the footplate and penetrated the area for about 0.5

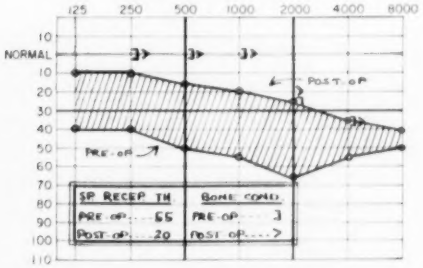

Fig. 4.—A 34-year-old woman with a history of bilateral progressive deafness for 12 years. Intermittent tinnitus in both ears. Preoperative hearing loss for pure tone and speech was 55 db. Following fenestration of the oval window through the center of the body of the footplate, hearing was improved to the level shown.

mm. with a clockwise and counterclockwise circular motion. When the explorer was removed, one could see into the dark vestibule through the circular opening. Perilymph was seen to escape from the fenestra of the oval window. Nine months after the operation, the hearing was at the 20 db. level by pure-tone and speech audiometry (Fig. 4).

The second type of stapedial obstruction causing conductive deafness results from thick inflammatory adhesions covering the entire footplate area and surrounding bone, thus preventing free movement of the footplate. This condition probably results from repeated recent or remote inflammatory or suppurative episodes. The eardrum in these cases may be intact or have a large or small permanent defect; the ossicular chain may be intact or in part destroyed. In other words, the stapedial deafness may be classified as chronic adhesive or chronic suppurative deafness.

In both these types of stapedial obstruction causing conductive deafness, fenestration of the oval window through the stapedial footplate has restored the hearing. Similarly, fenestration of the oval window through the footplate has restored hearing in unsuccessful Lempert fenestration cases.

In some cases of conduction deafness due to chronic suppurative disease, more of the edge of the posterior canal wall is removed, by either my method or the Tumarkin [4] technique of antrotomy, in order to deal with diseased tissue in the antrum and attic. Hearing has then been restored when the oval window was fenestrated, as in the following illustrative case. Thus in some cases, this procedure may achieve in a simpler manner the good results attained in the more complex tympanoplasty of Zöllner [5] and Wullstein.[6]

A locomotive engineer of 65 years of age had a history of deafness in both ears of 20 years' duration, following long-standing chronic bilateral suppuration, with intermittent discharge in both ears. For the past five years both the middle ears were dry. There was a constant humming tinnitus in the left ear. Both drums had a large marginal defect, including the posterior portion of Schrapnell's membranes, through which one could see what appeared to be a cholesteatoma membrane. There was no family history of deafness. The patient's preoperative hearing loss for pure-tone and speech was 40 db. in the left ear and 35 db. in the right.

On Oct. 17, 1955, by the technique employed in stapes mobilization, the contents of the middle ear on the left side were exposed. The bone of the posterior and superior canal wall was removed so that one could see into the attic and antrum. The incus was absent, almost all of the malleus was necrosed; what was left of it was removed, keeping the rest of the drum intact.

There was a great deal of thick, organized scar tissue in the tympanic cavity, which completely surrounded the stapes. There were also fibrous adhesions between the inner surface of the drum and promontory. When the adhesions around the stapes were removed, palpation of the stapes revealed that it was mobile and the hearing improved somewhat on the operating table. There was no evidence of otosclerosis. The round-window niche was freed of the scar tissue which filled it. After the footplate of the stapes was clearly defined, a fenestration of the oval window was made through the center of the footplate, through which one could see into the vestibule.

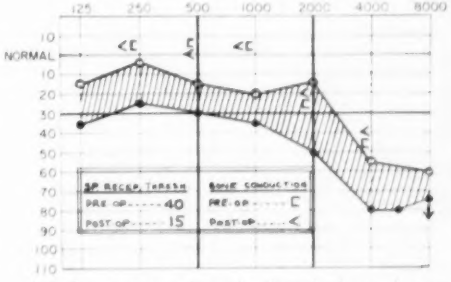

Fig. 5.—A case of conduction deafness due to chronic suppuration with marked hearing improvement following fenestration of the oval window.

Perilymph could be seen coming through the opening. After this maneuver, the patient's hearing improved to a marked degree on the table.

A portion of the skin removed from the anterior external auditory canal was placed over the region of the stapes and epitympanum, and the drum was replaced.

Ten months after fenestration of the oval window, the hearing was at the 15 db. level by speech audiometry (Fig. 5).

Summary

An effective instrument for fenestration of the oval window for otosclerotic deafness is presented.

Hearing can be restored in conduction deafness due to chronic adhesions or chronic suppuration by fenestrating the oval window through the footplate.

Oval-window fenestration therefore provides another pathway for physiological hearing.

101 E. 73rd St. (21).

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The Use of Meperidine in Peroral Endoscopies

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The growing number of peroral endoscopies necessitates the perfection of a convenient method of anesthesia or analgesia particularly suitable for these procedures. Intravenous thiopental (Pentothal) in combination with one of the muscle-relaxing agents is one of the best methods available. However, this combination necessitates the services of an experienced anesthetist and requires hospitalization of the patient. On the other hand, the usual local anesthesia alone, although sufficiently analgetic locally, frequently renders the examination very inconvenient for the patient. It is on account of these difficulties that physicians often hesitate to refer their patients for endoscopy as a routine procedure, especially in the case of non-hospitalized patients.

Ideally, the successful performance of endoscopies requires a general analgetic drug whose effect is of short duration, producing adequate muscle relaxation which at the same time leaves the patient sufficiently awake in order to cooperate, and which also has a wide margin of safety in order to be used routinely. Meperidine (ethyl 1-methyl-4-phenylpiperidine-4-carboxylate hydrochloride) seems to fulfill these requirements. Tradenames for Meperidine are Pethidine, Demerol, Dolestine, etc. The four carboxylate and four phenyl groups are apparently responsible for the analgesia produced.[1] The exact mechanism, however, of the analgetic action is, as yet, unknown. Goodman and Gilman[1] assume the analgetic effect to be due to the action of the drug on the cortical and subcortical regions of the brain as well as on the diencephalon. Meperidine also seems to act on striped and smooth muscle, although this effect of meperidine is experimentally difficult to prove.[1] On the whole meperidine relieves pain originating anywhere, but intestinal pain is more effectively suppressed than pain arising from nerves or bones.

The pharmacology of meperidine was extensively studied by Loeschcke et al.,[2] but its depressing action on the respiratory center is not yet clearly understood. Batterman and Himmelsbach[3] believe that in therapeutic doses meperidine can cause respiratory depression only in a few sensitive persons. Meperidine causes dizziness and vertical nystagmus, which originate in the vestibular nuclei.[4] Because of its rapid disintegration in the body it rarely causes toxic effects in therapeutic doses, and recovery from its general effect takes about two hours. It is, therefore, less dangerous than other analgetic drugs, such as morphine and its derivatives.

Because of these qualities meperidine seems to us to be a promising agent for the performance of peroral endoscopies. Since the middle of 1954 we have used it routinely in these examinations. In this paper we wish to report our experiences with the method.

Age Distribution of Patients

Age of Patients, Yr.	No. of Patients
15-20	10
21-40	78
41-60	91
61-82	71
Total	250

Accepted for publication July 23, 1956.

From the Ear, Nose and Throat Department of the Rambam Government Hospital.

Materials

Up to the end of 1955, we performed with the help of meperidine 250 endoscopies; 156 of these were bronchoscopies, 64 were esophagoscopies, and 30 were direct laryngoscopies. The age distribution of our patients is shown in the Table.

In the period covered we had no occasion to perform endoscopies in younger patients, but we would not hesitate to use it in children of over 4 years. In younger children we usually employ general anesthesia.

About one-third of the endoscopies were performed on ambulatory patients. The remainder were hospitalized patients referred for endoscopy by other departments of the hospital.

Our routine anesthetic procedure was as follows: About one hour before performing the endoscopy 1 mg. of atropine sulfate was administered subcutaneously to the patient. A sensitivity test for cocaine or tetracaine was carried out, by having the patient gargle 2 cc. of a 1% cocaine solution or 4 cc. of a 0.25% tetracaine solution. Anesthesia of the pharynx and larynx was then produced by instillation using 0.5% tetracaine solution in an amount of not more than 8 cc. or a 5% cocaine solution in an amount of not more than 5 cc. In the case of bronchoscopies a sufficient amount of cocaine and tetracaine solutions was left for the purpose of spraying the bronchi after introduction of the bronchoscope. After anesthetizing the pharynx and the larynx, meperidine was administered intravenously with the patient lying on the operating table. Immediately after completing the injection endoscopy was started.

The amount of meperidine necessary for an adult weighing 60-70 kg. is between 100 and 200 mg., using a 1% solution. Most of the patients received 150 mg., while six patients required 200 mg. Older patients usually required smaller amounts, whereas younger patients, whose general state of health was better, called for larger doses.

Results

Following the injection of meperidine a vertical nystagmus appeared, and the patients complained of dizziness. Their consciousness became slightly clouded, but they were able to answer questions correctly and to cooperate in the examination. Breathing became somewhat shallow and slower in rate. If the patient remained fully awake or the nystagmus did not appear, 20-50 mg. more of meperidine was administered before introducing the instrument; but it was also possible to add meperidine during the examination. The ambulatory patients remained under our observation for about two hours following the examination. After that they were able to find their way home, either with the help of a relative or often even on their own.

Comment

Intravenous meperidine, even without topical anesthesia, has been used for esophagoscopies and gastroscopies since 1951 [5,6] and recently also for suspension laryngoscopies.[7] Andersen et al.[8] were the first to note its advantages also for bronchoscopies. They state that with the help of intravenous meperidine endoscopic procedures become far less unpleasant for the patient, and the examiner enjoys having a patient who is undisturbed by anxiety and fully cooperative. Cimoch and Wirts[6] point to the advantage that the optimum effect of the drug is obtained at the exact time needed for the examination and does not depend on the time a premedication is administered in the ward. All observers agree in their favourable comment on the rapid onset of relaxation, even in nervous patients, which failed to appear only occasionally. No complications or side-effects apart from dizziness were observed if the drug was administered slowly and in the proper amount; only Andersen et al.[8] reported one case of severe but temporary respiratory depression, which was clearly due both to overdose and rapid injection of the meperidine.

In our experience the patient usually remains quiet and cooperative even if the examination requires time and is performed by a less skilled operator. A tense and apprehensive patient passes after the injection of meperidine into a mild state of euphoria. Most patients do not retain any unpleasant memories of the examination, and a number have partial amnesia of the procedure. In our hands the method also resulted in a

saving of staff, since only two nurses were needed to assist the operator. The muscle-relaxing effect of meperidine is particularly appreciated in esophagoscopies, the instrument passing easily through the cricopharyngeal sphincter. We were impressed by this muscle-relaxing effect in the performance of direct laryngoscopies and in those cases in particular where a thorough examination of the anterior commissure was required. Relaxation of the muscles of the neck was good even in subjects with a short and fat neck or when respiration was restricted by the presence of a laryngeal tumor. The danger of cocaine or tetracaine poisoning is considerably reduced by this method, as with meperidine even small quantities of these drugs give ample local anesthesia. In asthmatics meperidine is safe, is effective in suppressing the annoying cough, and is much less dangerous than morphine,[9] an effect which considerably facilitates bronchoscopies in these patients.

Prior to the introduction of this procedure we used to prepare patients for endoscopy with morphine. With the usual dose, however, many patients remained very apprehensive, while with larger doses they became uncooperative and unmanageable. The routine performance of endoscopies under general anesthesia met in our hospital with considerable difficulties, mainly on account of a shortage in trained anesthetists.

We have not seen any disadvantage resulting from the use of meperidine in the amounts mentioned, and no complications occurred. Intravenous meperidine can, however, cause an immediate and marked fall in the respiratory rate and tidal volume.[10] This respiratory depression can be caused by too much meperidine or by a too rapid rate of intravenous injection. It is therefore of the utmost importance to inject the drug very slowly, starting with a flow of 2-3 ml. per minute. The rate of injection can be increased only when after the first two minutes no ill effects on the central nervous system or on the respiratory system become apparent. It is advisable to have an instrument for positive-pressure breathing ready in the operating room. Nalorphine (N-allylnormorphine) is an antidote for meperidine.[11] It is a biological antidote, and not a chemical one, and is able to double or treble the rate of respiration. No case of respiratory depression occurred in our cases, although it was noted that the respiratory rate in most of our patients fell to six to eight per minute.

Summary

Intravenous meperidine in doses from 100-200 mg. was used as analgetic agent in peroral endoscopies in conjunction with local anesthesia. The advantages noted were muscular and psychic relaxation of patients who remained cooperative and reduction of amount of local anesthetic agent. No disadvantages were encountered in 250 procedures. The possible dangers of respiratory depression, which never occurred in this series, can be avoided by slow administration of the drug.

6 Melchett St.

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Otitic Complications

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The prechemotherapy era still remains vivid in the memory of the older physicians, particularly the otologists. Acute infections of the ear with mastoiditis and otitic complications were frequent, dreadful, and often tragic in their outcome. The patient suspected of having a complication of a middle-ear infection was examined carefully and repeatedly, both locally in the region of the involved ear and systemically, in order to detect early signs of spread beyond the middle ear. The otologist was skilled in ophthalmoscopy and neurologic diagnosis and utilized this knowledge.[1] Diagnostic measures, such as spinal taps, blood cultures,[2] Ottenberg differential blood cultures, cultures of ear discharge, and x-rays, were extensively used and their clinical implications clearly understood.[3,4] Surgery was the accepted method of treatment and when properly performed was a lifesaving measure.

The introduction of the sulfonamides in 1935 and the antibiotics in 1942 brought about a dramatic change in the concept of a hitherto unquestioned mode of therapy of ear infections. There was a remarkable reduction in the incidence of complications of middle-ear infections, and it seemed that those serious otitic complications—mastoiditis, sinus thrombosis, otitic meningitis, labyrinthitis, and brain abscess—would soon be only of historical interest.[5] In the first flush of enthusiastic treatment, every patient treated by the general practitioner, the pediatrician, and the otologist was cured. There seemed to be no further need for that fine clinical observation and evaluation of symptomatology evolved after years of experience by the otologist. The successful treatment of middle-ear infections seemed to have become entirely nonsurgical.

In the past few years, however, a gradual change has begun to become evident. Complications of middle-ear infections are beginning to be seen at otologic centers throughout the country in increasing numbers.[6,7] These complications again carry with them the same threat to life and function as noted in the era preceding the antimicrobial agent. The complacency of the general practitioner and the pediatrician, who were treating middle-ear infections, no longer seems warranted, and is being replaced by a sense of uneasiness. A series of complications of ear infections recently observed at The Mount Sinai Hospital attests to the continued importance and seriousness of this type of infection.

Report of Cases

CASE 1. (acute mastoiditis with a perisinuous and an extradural abscess).—A 5½-year-old boy entered the hospital because of intermittent fever of seven weeks' duration following a mild upper respiratory infection. He had been treated at the onset with small doses of penicillin orally, followed by a sulfonamide for a few days, with subsidence of fever. A few days after medication was stopped, there was a recurrence of fever. He was thoroughly worked up at this time by a pediatrician with completely negative findings, except for a positive tuberculin test. Again he was placed on medication, consisting in turn of oxytetracycline (Terramycin), chlortetracycline (Aureomycin), and then chloramphenicol (Chloromycetin). Each time there would be an apparent clinical improvement with a subsidence of the temperature but only to be followed by recurrence of fever when medication was stopped.

Examination on admission revealed a somewhat ill-appearing youngster, with no particular complaints; the temperature was 102 F and the pulse rate 104. Abnormal physical findings were limited to the right ear. The right eardrum was flat

Accepted for publication Aug. 27, 1956.

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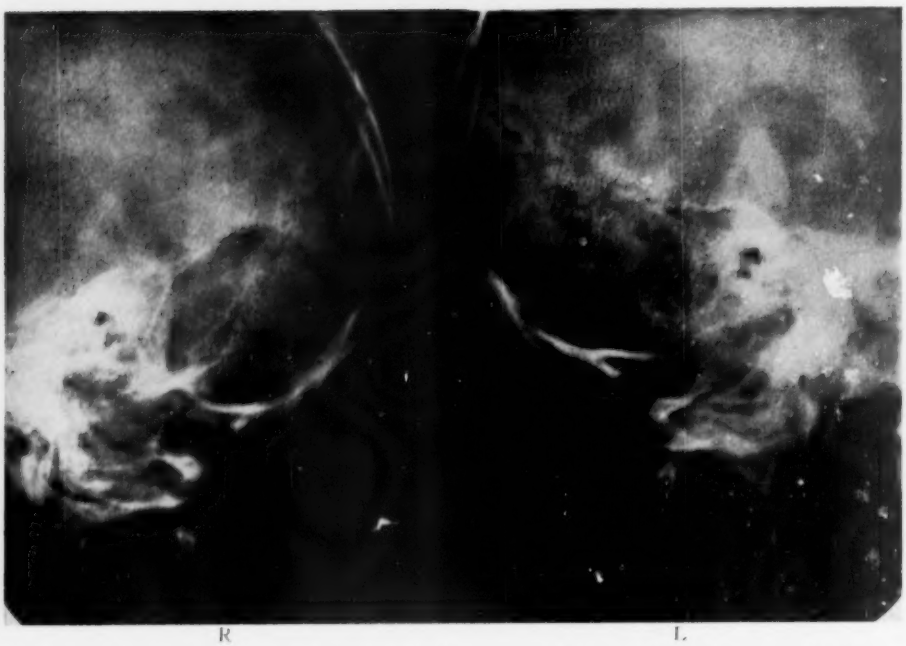

Fig. 1.—The findings of the roentgenologist were as follows: "Extensive pneumatization of both mastoids with a diffuse clouding of all the cells on the right side. There was no evidence of any localized area of bone destruction. The sinus plate appeared distinct as did the superior margin of the petrous pyramid."

though somewhat thickened. Normal landmarks were present. There was definite bony tenderness far back over the region of the lateral sinus. X-rays taken at this time (Fig. 1) revealed extensive pneumatization of both mastoids, with a diffuse clouding of all the cells on the right side. This was interpreted as showing no evidence of any localized area of bone destruction. The sinus plate appeared distinct, as did the superior margin of the petrous pyramid. When on antibiotic agents, patients with such findings must be viewed with altered criteria as to the significance of such roentgen findings. Operation was performed several hours after admission. A postauricular incision was made. On removal of the cortex, there was extensive breaking down of the mastoid cells throughout, with granulations in the initial groove, tip cells, and the cells behind the sinus. Several cubic centimeters of creamy yellow pus issued from a mass of granulations over an exposed lateral sinus, which region corresponded precisely to the area of preoperative bony tenderness. The perisinuous abscess was widely exposed. It was necessary to make a posterior incision to encompass the extent of the disease posteriorly. The infection also extended up on the dura for about 4 cm. The diseased area was widely exposed until the normal-looking sinus was reached, both at the bulb and toward the torcular. Likewise, the cerebellar and the middle fossa dura were exposed until healthy-looking dura was seen.

The child's postoperative course was entirely uneventful. Antibiotics were deliberately withheld so as not to obscure clinical findings which might point to involvement of the lateral sinus. The temperature, taken every two hours, returned to normal within three days and remained thus for the rest of the patient's two-week convalescent period, during which time the wound healed completely.

Culture of the pus from the mastoid revealed a beta hemolytic Streptococcus. Sensitivity tests revealed this organism to be twice as resistant as the standard to penicillin, 20 times as resistant to chlortetracycline, and 100 times as resistant to sulfadiazine. Pathological examination of the bone and soft tissue removed at operation revealed acute nonspecific inflammation.

Comment.—A destructive mastoiditis with a large perisinuous and an extradural abscess caused by a penicillin-sensitive Streptococcus was present with a relatively normal eardrum. There were minimal clinical symptoms and signs of mastoiditis. The

sensitivity tests indicated that the only medication which might have been of value at the onset of the infection was penicillin. In this instance penicillin had been given only in small amounts and orally rather than by injection.

CASE 2.—A 52-year-old man first became ill eight weeks prior to admission, at which time he experienced pain in the left ear. He received several injections of penicillin during the next few days, the left eardrum being red. The ear pain slowly subsided, but because of some persisting right-sided headache, x-rays of the skull and mastoids were taken, which were reported as showing no roentgen evidence of disease. He felt relatively well until four weeks prior to admission, at which time he became dizzy and nauseated and noted some impairment of hearing on the left side. He was seen by several physicians, all of whom regarded the symptoms as those of Ménière's disease. He was treated with dimenhydrinate (Dramamine), with some relief of symptoms. X-rays of the mastoids were repeated and were reported again as showing no evidence of disease (Fig. 2). Five days prior to admission the pain in the left ear recurred, with increasing radiation of the pain to the eye, left temple, and teeth. He was first seen by one of us (H. R.) at this time at home; evidence of meningitis was noted and the patient was promptly hospitalized.

On admission, examination revealed an acutely ill, well-oriented patient, complaining of left-sided headache. The temperature was 102 F and the pulse rate 84. Moderate neck rigidity was present. The left eardrum was thickened, red, and without landmarks. No hearing could be elicited in this ear with the Bárány noise apparatus in the right ear. The mastoid region was not tender; no canal wall change was noted. The right ear appeared normal. There was a coarse nystagmus to the right. The blood study at this time was as follows: hemoglobin content 12 gm/100 cc., white blood cell count 13,250, with 80% polymorphonuclear leucocytes, 17% lymphocytes, and 3% monocytes.

A spinal puncture done at this time revealed cloudy fluid, with a pressure of 270 mm., containing 1190 cells, 52% lymphocytes and 48% polymorphonuclear leucocytes, a protein level of 118 mg/100 cc., and no sugar. There was no growth from this fluid.

Therapy was promptly started, consisting of procaine penicillin G, 600,000 units; streptomycin sulfate, 1 gm. per day, and crystalline penicillin, 300,000 units every three hours. The patient's general condition rapidly improved. Spinal tap performed four days after admission revealed a

Fig. 2.—X-rays of the mastoids were reported as "showing no evidence of disease." However, the faint defect in region of zygoma in a patient on heavy antibiotic treatment should alert one to potential area of destruction as the cause of meningitis.

clear fluid with 57 cells, 20% polymorphonuclear leucocytes, 80% lymphocytes, a protein level of 65 mg/100 cc., and a sugar level of 70 mg/100 cc. The vertigo as well as the left-sided headaches persisted during the next few days; the temperature remained at normal levels.

One week after admission, a left mastoidectomy was performed through a postauricular incision. A large cellular mastoid was extensively diseased throughout, containing stringy mucoid exudate suggestive of the Type 3 Pneumococcus infections frequently seen in former years. The disease involved all the perilabyrinthine cellular bone, extending into the petrous pyramid. The middle fossa plate was widely taken down; no extradural abscess was noted. The posterior bony canal was taken down, revealing extensive granulations encasing the incus and malleus. The incus and the granulations were removed. There was necrosis of the labyrinthine capsule of the external semicircular canal with granulations protruding from the interior of the labyrinth. The tegmen in the tympanic and epitympanic area was absent; there were granulations over the dura at this point just anterior to the ampullated end of the external and superior semicircular canals and just posterior to the geniculate ganglion. No organisms grew from the culture of the material from the mastoid.

The patient's postoperative course was uneventful. He was continued on procaine penicillin G for one week. This medication was gradually decreased, then stopped by the end of the second week. Patient was asymptomatic, except for persisting deafness in the left ear. He was discharged three weeks after the operation, the wound having completely healed.

Comment.—This patient was seen by several physicians, otolaryngologists, internists, and a neurologist, all of whom were unimpressed by only mild changes in the eardrum and apparently were reassured by the normal x-ray reports. Later review of the x-rays still showed only minimal clouding. There have been other instances in which the use of antibiotics significantly altered the expected x-ray changes, an almost normal appearance being maintained. Again noteworthy is the fact that no identifiable organism was produced from culture of the obviously diseased bone and secretion recovered from the mastoid.

CASE 3.—A 7-year-old boy had an earache on the right six weeks before admission. He was treated with penicillin by mouth for three days, and seemed to be well, except that the hearing in the right ear appeared impaired. Two weeks later, he again became ill with recurrence of the earache on the right and a temperature rise to 107 F. A diagnosis of "flu" and bilateral catarrhal otitis media was made. He received one injection of penicillin followed by oral penicillin for the next two days. Again he appeared well and remained so until four days before admission, at which time there was recurrence of the earache, now associated with vomiting and a temperature of 103.6 F. Again the diagnosis of bilateral catarrhal otitis was made, and the patient received an injection of penicillin. The next day the temperature was normal and the earache had subsided; however, the vomiting persisted and the child began to complain of frontal headaches and dizziness. On the day preceding admission, he began to have diplopia. He was first seen at the time by the pediatric consultant, who promptly referred the child to our hospital, with the tentative diagnosis of otitic meningitis.

Examination revealed a moderately ill-appearing youngster who seemed alert and well oriented. The Kernig sign was positive; the ocular fundi appeared normal. There was a paresis of the right sixth cranial nerve. The right eardrum appeared somewhat dull but not reddened. No other abnormal findings were noted. The temperature was 100 F and the pulse rate 60. A spinal tap revealed clear fluid under normal pressure. Lymphocytes numbered 3, sugar level was 80 mg/100 cc., and total protein content was 12.3 mg/100 cc. No organisms were seen on direct smear, and culture of the fluid yielded no growth. The blood study was as follows: hemoglobin content 14 gm/100 cc., white blood cell count 9800, polymorphonuclear leucocytes 56, lymphocytes 29, and mononuclear leucocytes 5. X-rays of the mastoids revealed "A diffuse haziness overlaying the entire right mastoid region, obliterating the intercellular septa and obscuring the pneumatic structures; there was no definite evidence of bone destruction" (Fig. 3).

The attending pediatrician decided to treat the patient with antibiotics; accordingly, he was started on penicillin, 1,200,000 units, and chloramphenicol, 1 gm. per day. The patient was first seen by one of us on the day after admission, at which time the right eardrum was dull, with no light reflex. Tenderness was noted over the right mastoid region. Immediate surgery was advised. However, the pediatrician wished to give the patient a further trial with medical management. Two days later the child was again seen by the otologist, at which time the child had right hemicranial headache and appeared very ill. Operation was now promptly done.

A postauricular incision was made; the mastoid cortex was exposed, the bone bleeding on exposure. The cortex was removed; all the cell

Fig. 3.—X-rays of mastoids reveal "diffuse haziness overlaying the entire right mastoid region obliterating the intercellular septa and obscuring the pneumatic structures. There was no definite evidence of destruction." In view of the history of prolonged antibiotic therapy, conclusion must not be drawn that there is no evidence of destruction.

walls appeared soft and necrotic. In the region of the tip there was extensive necrosis of the cells with necrotic granulations. The lateral sinus plate was necrotic. There was an extensive perisinuous abscess with organized blood clot between the sinus plate and the sinus wall. The sinus was exposed from the jugular bulb to 1 in. behind the knee until healthy dura was seen. The semicircular canals were skeletonized and leads into the petrosa followed until healthy bone was encountered. The middle fossa dura was exposed in order to inspect the superior surface of the petrous pyramid; no extradural abscess was noted.

The patient's postoperative course was entirely uneventful. There was a rapid disappearance of the signs of meningeal irritation. The paralysis of the right external rectus cleared in two days.

The culture of the material taken from the mastoid cavity at the time of operation failed to produce any organisms. The operative wound was entirely normal, and the drum and hearing appeared normal at the time of discharge from the hospital, two weeks following the operation.

Comment.—We were dealing here with a child who had an innocuous-looking middle ear which never had discharged. He had meningeal irritation, secondary to mastoiditis with perisinuous abscess, which became evident after six weeks of intermittent, inadequate antibiotic therapy. Also noteworthy is the failure to obtain a growth from the diseased material removed at operation. This child was most fortunate in that he did not develop a thrombosis of the lateral sinus.

Case 4.—A 19-year-old boy had a chronic discharge from the left ear for seven years, with no associated symptoms and no pain. Three weeks before admission to our hospital he noted a pain in the left ear region, persisting for the next week, at which time a severe throbbing left frontal headache, nausea, and vomiting were noted. He was hospitalized elsewhere. Examination at that time revealed an acutely ill patient with an obvious meningitis. Spinal fluid culture revealed Proteus vulgaris. He received large amounts of oxytetracycline, with a gradual decrease in the originally high temperature but with a persistently positive spinal fluid culture for P. vulgaris. After two weeks, during which time there was a rapid deterioration in his general condition, he was seen for the first time by an otologist who recognized the significance of the ear infection in rela-

tion to the persisting meningitis and arranged for transfer of the patient to our hospital.

Physical examination on admission here revealed an acutely ill patient in marked distress, complaining of severe right-sided temporal headache. Neurological examination revealed positive Kernig and Brudzinski signs, generalized hyperactivity, with no other abnormal findings or localizing signs. The right ear appeared normal. There was an almost total defect, marginal, of the left eardrum, with a small amount of malodorous secretion in the middle ear. The hearing was moderately impaired. No bony tenderness was elicited. There was no nystagmus or eye muscle palsy. The eye grounds appeared normal.

In view of the presence of the persisting meningitis in this critically ill patient, and because of the lack of any localizing intracerebral signs, a mastoidectomy was immediately scheduled. Anesthesia was started by mask, using a nitrous oxide-oxygen-ether mixture. After a few minutes the patient suddenly developed a complete respiratory standstill, with maintenance of normal cardiovascular function. He was placed in a respirator after attempts at respiratory resuscitation proved unsuccessful. A neurosurgical consultant saw the patient at this time and found no evidence of increased intracranial pressure. The patient was comatose with fixed dilated pupils and areflexic.

Eighteen hours later, during which time there was no change in the patient's condition, it was decided to explore the mastoid. An intratracheal tube was introduced, through which artificial respiration was maintained and the respirator could be stopped. A postauricular incision was employed; a large infected cholesteatoma of the antrum and aditus was found, with a tremendous perisinuous abscess extending from the sinodural angle to the bulb, and an extradural abscess extending posteriorly over the cerebellum. The neurosurgeon witnessing the operation did not feel that the underlying cerebellum was abnormal. There was no change in the patient's condition after the operation, and he died the next day.

Postmortem examination revealed a very deeply situated cerebellar abscess containing foul pus.

Comment.—In brief, we were dealing with an infected cholesteatoma with complicating perisinuous, extradural, and cerebellar abscesses. In retrospect, the errors in the care of this patient are evident. There was laxity during the seven-year period of chronic middle-ear suppuration in appreciating the potential danger of this type of ear infection. Secondly, there was a startling lack of awareness on the part of the attending physicians in the first hospital in failing to recognize promptly that this was an otitic meningitis. Thirdly, there was apparent unconcern with the fact that the infective agent was P. vulgaris, which is notoriously resistant to almost all known antibiotic agents and requires proper surgery for its eradication. Fourthly, there was the error of omission on the part of the otologic surgeon here to aspirate the cerebellum at the time of the mastoidectomy.

Our recent experiences parallel those of others. There are at least three aspects to this problem which merit discussion: (1) the reasons for the continued development of otitic complications, (2) the increased difficulty in making an early diagnosis, and (3) the lack of awareness on the part of the physicians who treat the great bulk of the early otitic infections of the continued development of otitic complications.

1. The Continued Development of Complications

The varied and serious complications of middle-ear infection were thoroughly elucidated by the otologists in the prechemotherapy era and need not be repeated here.[8] In our experience, the organisms responsible for the infections and their complications were, in order of frequency, the beta hemolytic Streptococcus, the various strains of Pneumococcus (particularly Type 3), the Staphylococcus aureus, the Friedländer bacillus, the Proteus vulgaris, and the Bacillus pyocyaneus. The types of organisms causing middle-ear infections have remained basically unchanged; likewise, the types of complication are similar to those seen in the prechemotherapy era.

Our experience, which is in accord with the reported experiences of others, indicates that there may be one of several factors, alone or in combination, responsible for failure of the commonly employed antimicrobial agents to eradicate the ear infections in the early stages. Basic is the obvious fact that the offending organism must be sensitive to the action of the medi-

cation being used. Fortunately, the vast majority of acute otitic infections are caused by organisms susceptible to broad-spectrum antibiotics. There has been noted with increasing frequency the presence of strains of organisms which are resistant to the action of the previously effective medication. This is most striking at present among the strains of the staphylococci, a substantial number of which are now resistant to penicillin.[9] The mechanism for the development of these resistant strains has been discussed by others.[10,11] The clinical importance of this phenomenon is, of course, obvious. Shifting empirically to another antibiotic is not always the panacea, because not infrequently multiple resistance to other antibiotics now occurs with increasing frequency.

Accordingly, the early identification of the organism by bacteriologic methods and the determination of its sensitivity become increasingly important, especially in what appears to be a refractory infection. The taking of a culture of an ear discharge presents no difficulties. In those instances in which we are dealing with a patient with an intact drum myringotomy or aspiration for culture purposes appears to be an entirely logical, justifiable procedure. To us, it appears greatly to be preferred to the haphazard shifting from one antibiotic to another in a trial-and-error fashion, during which time an uncontrolled infection may spread and an otitic complication be initiated. This method will, in our opinion, lessen the tendency for bacterial resistance to develop.

In addition to using the proper medication, i. e., one to which the offending organism is susceptible, the medication must be given in sufficiently large doses to be effective, and for a sufficient length of time to eradicate the infection. There are as yet no firm and fast rules which determine the amount and duration of treatment. This is where mature clinical judgment and understanding of the nature and extent of the infection aid in the decision. In the case of the sulfonamides, a fairly firm relationship exists between the blood levels attained and the effectiveness of the medication and the optimum dosage for known organisms have been established. The use of the antibiotics, especially penicillin, has been much more empiric, with an increasing tendency to use long-acting forms and oral preparations, which we decry. While these forms are or may at times be adequate, it has been our experience repeatedly to see patients who were not doing well but who promptly began to improve when the amount of penicillin was sharply increased. On a few occasions, the use of crystalline penicillin in large doses every few hours has proved lifesaving in what otherwise appeared to be a penicillin-resistant infection.[11] (Davison likewise advocates the use of substantially larger amounts of penicillin in the routine treatment of an acute middle-ear infection.) The failure of organisms to grow (which are seen on direct smear) is very likely an indication at times of the only partial effectiveness of the medication due to inadequate dosage. However, the impression should not be left that dosage alone is the key to successful treatment; a resistant strain will not respond regardless of the dose.[12]

To obtain a satisfactory therapeutic result with an antibiotic to which the bacteria are sensitive, it is necessary that the medication reach the bacteria in adequate concentration. This basic fact has emerged repeatedly in the treatment of infections in all parts of the body: subacute bacterial endocarditis, obliterative lateral sinus thrombosis, etc. Once there is a definite collection of pus, the effectiveness of a theoretically useful medication is greatly obtunded by its inability to penetrate so that the bacteriostatic or bacteriocidal action cannot be brought into play. Elementary surgical principles applied without hesitation in other parts of the body would indicate the necessity for establishing, under these circumstances, surgical drainage. The efficacy of such a procedure is manifest;

the reluctance on the part of some to have this applied in the case of otitic infections is difficult to understand. An infection which involves bony structures likewise presents a problem of penetration of the medication. Once a definite bony focus has been established, surgical drainage and removal of the diseased bone is indicated. We are of the opinion that none are so well qualified as the otologist to make this decision. Delay in instituting surgical drainage may result in the development of an otitic complication. We know this to be true clinically because of the proportionally greater increase in otitic complications in relation to the far fewer cases of mastoiditis which occur.

At the risk of repeating the obvious, it should again be stressed that adequate amounts of the effective medications should be used; cultures obtained; sensitivity tests performed, and the antibiotic of choice should be continued for a sufficient length of time, certainly until all evidence of inflammation has subsided.

2. Increased Difficulty in Making an Early Diagnosis of a Complication of an Otitic Infection

The experienced otologist of the prechemotherapy era knew that not all complications of otitic infections presented the classic clinical picture and typical laboratory findings. Otitic complications caused by the Pneumococcus Type 3 ran a characteristically insidious clinical course. The patient's symptoms might consist of slight stuffiness in the ear or tinnitus; there might never have been an ear discharge, merely slight thickening or minimal inflammatory change. Bone tenderness was usually absent or hard to elicit. The blood studies were of little value. The temperature usually revealed little deviation from normal. The x-ray, however, revealed a pneumatized mastoid with a thick cortex, explaining the absence of mastoid tenderness. Diffuse haziness was a rather common finding. Operation, however, would often reveal an extensively destructive process in the mastoid and perilabyrinthine cell structure. Otologists of experience suspected this type of dangerous mastoiditis and by timely surgery were able to cure such patients in the premedication era.

The typical picture seen in otitic infections modified by the use of antimicrobial agents strongly resembles the picture of Type 3 Pneumococcus.[13] The early use of antimicrobial agents often seems to suppress the clinical signs and symptoms of advancing infection in the ear and temporal bone, slowing it down but not checking its spread. Accordingly, one must be increasingly on the alert for the undramatic symptoms and findings which at present may be the only evidence of widespread underlying disease. Persistence of any inflammatory changes in the drum, persisting impaired hearing, slight temperature, spontaneous headache, vertigo following an ear infection even in the presence of a relatively normal-appearing drum, genuine bony tenderness—any one or a combination of these must alert the responsible physician to search for an explanation.

There cannot be, nor was there ever in the past, too great a dependence upon laboratory findings. When laboratory data confirm clinical impressions the experienced will accept these findings, but when the data are not in accord with clinical impressions it is wiser that they be viewed most critically. It is noted in increasing instances that in patients treated with antibiotics for some time the x-rays may be reported as normal or showing very slight clouding. At operation, one does not usually find the large coalescent abscess within the mastoid process, but rather multiple areas of infection with softening of the bony cell margins, but without the disolution of these bony septa. X-rays may fail to reveal, accordingly, the intensity or extent of the infection, since the intercellular septa may still be seen.

We must, accordingly, revise our criteria for suspecting mastoiditis and be highly

suspicious of any local symptom or clinical finding in a patient with a history of recent otitic infection treated for any protracted time with an antimicrobial agent.

3. Lack of Awareness of the Continued Existence of Otitic Complications

The great bulk of early otitic infections are being treated by family physicians and pediatricians. This is as it should be. The widespread use of the sulfonamides and the antibiotics in these acute infections[14] has so changed the over-all outlook in these infections as to justify the term "miracle drugs." The effect of the antimicrobial agents in the treatment of chronic ear infections has been nowhere near as dramatic.

Interestingly enough, physicians recognize the limitations of these drugs in chronic infections and usually are less reluctant to obtain the opinion of the otologist for this type of problem. The conscientious physician must be on his guard to avoid becoming complacent in treating ear infections. From all over the country come reports that "atypical" mastoid infections are being seen in increasing numbers. It is important to alert physicians to the continued importance and danger of this type of infection. More difficult to understand is the almost dogmatic resistance of some experienced physicians to the fact that otitic complications are still occurring and require surgical intervention for their cure. Modern temporal bone surgery concerns itself not merely with the elimination of infection and saving of life but also with the preservation of function.

Summary

Otitic complications are occurring with increasing frequency, particularly in proportion to the number of cases of mastoiditis encountered.

The factors which have some bearing on the apparent increased incidence of otitic complications may be bacterial resistance, inadequate dosage in early stages of infection, unscientific choice of proper antimicrobial agent, and discontinuance too soon of an effective agent based on defervescence of symptoms rather than complete subsidence of all signs and symptoms of the infections.

It is of importance to emphasize the frequency with which otitic infections treated inadequately or incompletely with antimicrobial agents tend to simulate the clinical picture of the mastoiditis caused by Type 3 Pneumococcus.

Otitic complications, i. e., mastoiditis with perisinuous or epidural abscess, sinus thrombosis, brain abscess, labyrinthine capsule necrosis, facial paralysis coming on late after the onset of an acute ear infection, are unquestioned surgical emergencies. The best interests of the patient will be served by proper surgery. The utilization of the appropriate antimicrobial agent after the suppurative focus has been eradicated will greatly aid in reducing mortality and morbidity.

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Personal Experiences with Stapes Mobilization

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The surgical treatment of clinical otosclerosis by mobilization of the stapes footplate is not a new procedure. Kessel [1] as early as 1876 recognized footplate ankylosis and made surgical attempts to create a movable footplate. He was followed by Miot,[2] Blake,[3] Jack,[4] and many others. Except for sporadic attempts, the procedure was largely discontinued about the turn of the century, apparently due to unsatisfactory and disappointing results.

When one recalls these otologists had rather crude instruments, inadequate anesthesia, improper illumination and magnification, total lack of electronic testing devices, and no chemotherapeutic or antibiotic drugs, the reasons for the failure of the procedure in those early attempts become quite apparent.

Dr. Julius Lempert [5] described an approach to the middle-ear area for tympanosympathectomy that allowed exploration of the middle ear with relative ease.

Dr. Samuel Rosen [6] used the basic exploratory approach of Lempert to revive the procedure of stapes mobilization. Largely through his efforts this procedure has now found a permanent place in otologic surgery for the treatment of otosclerosis.

The further writings of Rosen,[7] Goodhill,[8] Kos,[9] Bellucci,[10] and Scheer [11] and the work of Kenneth Day,[12] P. Meltzer,[12] G. Shambaugh, Jr.,[12] T. Sooy,[12] S. Baron,[12] S. Derlacki,[12] John Shea,[12] John Rambo,[12] H. Wullstein,[12] and many others are well known to those of us active in mobilization surgery.

Accepted for publication Sept. 30, 1956.

From the University of Southern California School of Medicine at Los Angeles.

Presented at the Annual Meeting of the Pacific Coast Oto-Ophthalmological Society, Phoenix, Ariz., in April, 1956.

Personal Observations

Some two years ago I had the pleasure of observing the work of Dr. Rosen. I was very pleased with my observations. The anesthetic and surgical approach were fascinating to observe. It was apparent that stapes mobilization with Rosen's original technique of posterior pressure on the neck of the stapes did improve hearing to the serviceable level and maintained that improvement for at least two years in some cases.

Stapes Mobilization Performed Simultaneously with Fenestration Surgery

On my return to Los Angeles I began mobilizing the stapes footplate according to the Rosen technique routinely during fenestration surgery. This afforded an ideal exposure of the stapes footplate area, both before and after mobilization. After using this technique on some 50 consecutive fenestration cases several conclusions were obvious, as follows:

1. Mobilization of the footplate performed simultaneously with fenestration in no way altered the results of the fenestration procedure.

2. Under ideal visual exposure posterior pressure on the stapes neck, in my experience, resulted in crural fracture in some 50% of the cases.

Figure 1

Fig. 2A.—Fixed footplate.

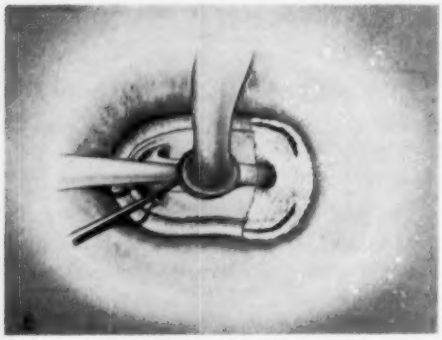


Fig. 2B.—Gentle superior-inferior and oblique motions produced a marginal fracture in the longitudinal axis of the fixed footplate.

3. I soon observed that the convex curve of a dulled needle placed gently on the edge of the capitulum could be used for stapes mobilization.

4. It was likewise noted that slight pressure applied superiorly and inferiorly resulted in motion of the superior and inferior margins of the footplate. Slight oblique pressure seemed to increase this footplate movement.

After freeing the longitudinal axis of the footplate, the remaining small amount of anterior footplate fixation could then be fractured with greater ease by applying gentle pulsating pressure in direct line with the posterior crus.

The development of a slight posterior tilt indicates the completion of the fracture through the otosclerotic process anteriorly.

A more marked posterior tilt seemed to indicate a tear in the annular ligament, as evidenced by increased bleeding about the footplate margin, as well as increased mobility.

Occasionally, a transverse fracture in the middle one-third of the footplate would occur with simultaneous fracture of the anterior crus (Fowler anterior crurotomy[13]).

In some instances pressure on the capitulum caused the neck of the stapes to split. For these cases a small capitulum cup was designed to fit over the entire stapes head through which manipulating motions could be carried out.

Direct Footplate Manipulation

In spite of these efforts and methods some footplates could not be mobilized without crural fracture. When excessive pressure seemed necessary and fracture of the crura appeared imminent, or if fracture of the crura did occur, obviously a direct approach to the footplate was essential if the

Fig. 3.—Application of pulsating pressure in direct alignment with the posterior crus completes the fracture.

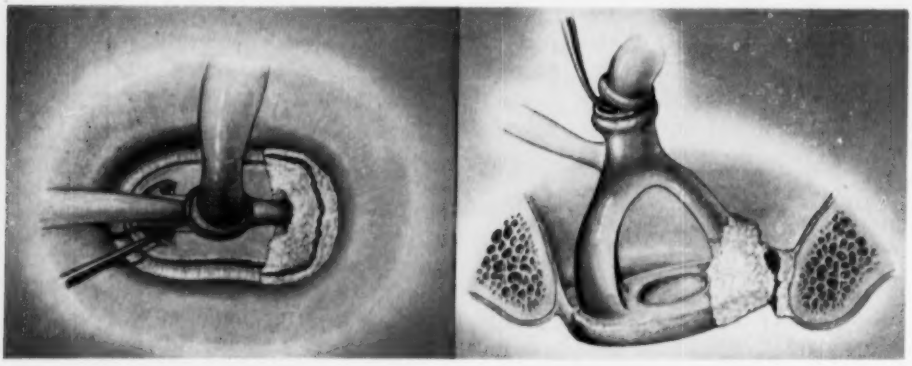


Fig. 4A.—Normal footplate position.

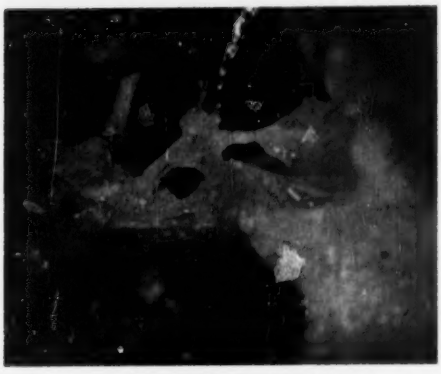


Fig. 4B.—Moderate tilting without ligament tear.

Fig. 4C.—Marked tilting with ligament tear.

conduction block due to a fixed footplate was to be eliminated. In these cases direct needle manipulation was attempted and if mobilization could not be achieved, the pneumatic hammer technique was applied.

Direct Needle Manipulation of the Fixed Footplate

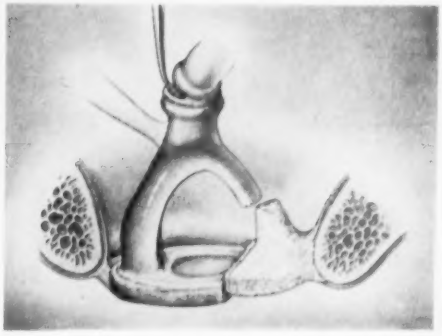
Some footplates that could not be mobilized through the crura can be freed or fractured by applying pressures through a dulled needle placed along the footplate margins. The most vulnerable area seemed to be in the posterior one-half of the footplate, especially in the region of the attachment of the posterior crus to the footplate. After fracture of the footplate occurred by direct needle application, the posterior tilting motions could often be improved by then returning to the capitulum of the stapes and applying a posterior pulsating pressure, providing crura were intact.

The Pneumatic Hammer Technique

Manual needle manipulation of the footplate area is not without certain hazards. If considerable pressure is applied and the footplate suddenly fractures, there is a tendency for the needle to drop toward the vestibule before the manual pressure can be released.

Recognizing this hazard in direct footplate manipulation by manual means, I developed a modified dental pneumatic hammer in collaboration with Mr. Glenn Turner of Los Angeles. The principle involved was to create a controlled pulsating pressure that can be varied as desired, as to both intensity

Fig. 5.—Transverse footplate fracture with simultaneous fracture of the anterior crus (Fowler anterior crurotomy).



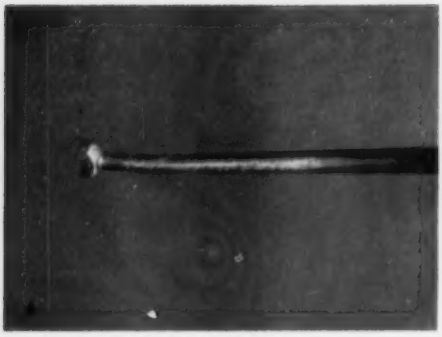


Fig. 6A.—Capitulum cup.

Fig. 6B.—Capitulum cup in mobilizing position.

and frequency. This hammer considerably lessens the hazard of dropping toward the vestibule after sudden fracture of the footplate occurs.

With the use of controlled pulsating pressures, as delivered through the pneumatic hammer, very few footplates were found that could not be fractured transversely or at their margins. In some cases a shattering effect was noted.

Figure 7

In the event of crural fracture, footplate manipulation, either manually or by the pneumatic hammer, or both, is justified, since the posterior crus often seems to reunite with the footplate. The anterior crus does not seem to heal after fracture. This may be due to the fracture sites being in contact with the otosclerotic bone. These observations have been made during fenestration surgery on cases that had had a previous stapes mobilization performed.

In general, direct footplate manipulation by any means is somewhat hazardous. If mobilization of the stapes and footplate as a unit can be accomplished by pressures applied through the crura, it is not only safer but also seems to give the best over-all result, both in hearing gain and permanency.

Present Technique

Preliminary Procedure.—A preoperative air-conduction audiogram is made in the operating room using the compact portable

Fig. 8A.—Pneumatic hammer, motor, and foot switch.

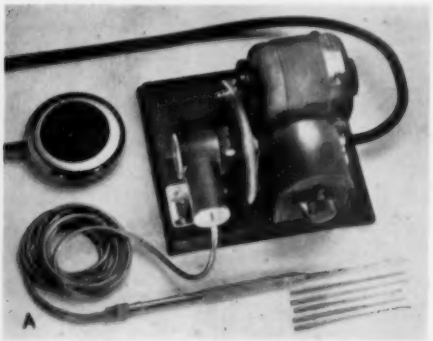
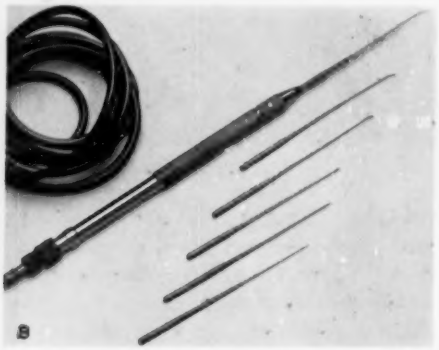


Fig. 8B.—The pneumatic hammer with various hammer points.



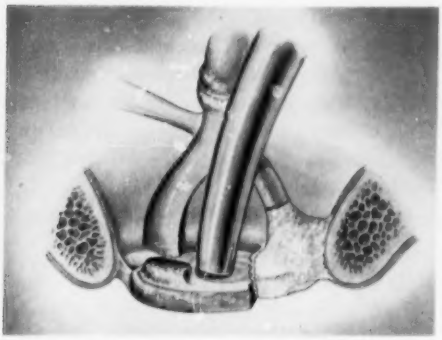


Fig. 9.—Hammer point in position on the footplate.

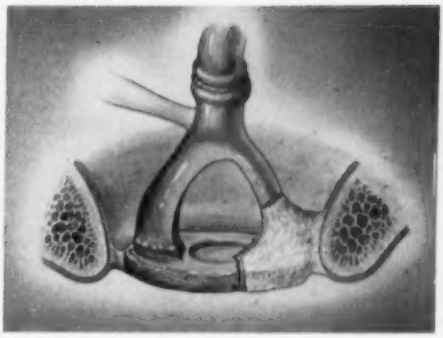


Fig. 10.—Note the healed posterior crus and the nonunion of the footplate and the anterior crural fracture.

otometer specifically designed for this purpose.

Preoperative Anesthesia.—Morphine, ⅛ grain (8 mg.), is given one-half hour before surgery. Any additional sedation would dull the patient's ability to respond accurately to the hearing tests performed at the time of surgery.

Reassurance is continually given to the patient immediately before and during the surgical procedure to lessen the anxiety.

Operative Anesthesia.—Approximately 3 cc. of 2% lidocaine (Xylocaine) and 1:1000 epinephrine (6 parts to 1) is infiltrated subcutaneously about the external circumference of the external ear canal.

Incision.—Vertical incisions are made superiorly and inferiorly, beginning 1 mm. from the eardrum margin and extending outwardly approximately 0.5 cm. These two incisions are then connected transversely. This incision provides an adequate skin flap with a minimum of bleeding.

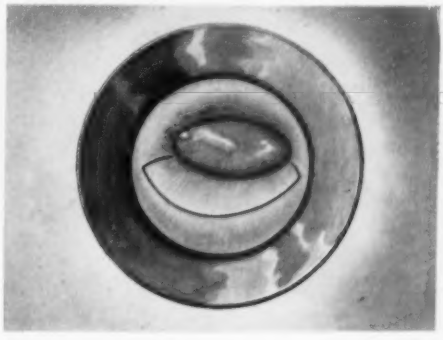
Separators are used to elevate the skin to the annulus. Any oozing which may occur is controlled by pressure with cotton applicators dipped in 1:1000 epinephrine solution.

Exposure at Middle-Ear Area.—A small elevator enters the notch of Rivinus and the eardrum is carefully elevated from its sulcus, exposing the posterior one-half of the middle ear. If the chorda tympani nerve obscures the incudostapedial area, it may be stretched inferiorly or severed.

A modified Fowler curet is used to remove any bony overhang sufficient to visualize the pyramidal process. This exposure allows visualization of the following structures:

Fig. 11.—Otometer.

Fig. 12.—Outline of incisions.



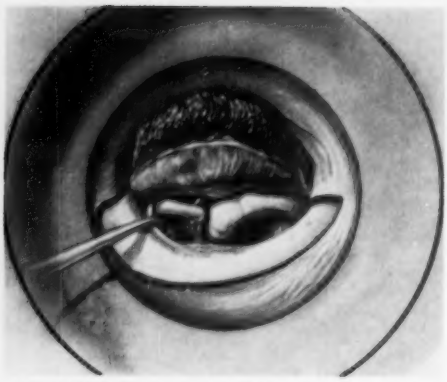


Fig. 13.—Exposure of middle-ear area.

1. Transverse portion of the Fallopian canal.
2. Long process of the incus and the incudostapedial joint.
3. The stapedial tendon and the pyramidal process.
4. The posterior crus and often the anterior crus of the stapes.
5. The footplate and its superior or inferior margins, or both.
6. The promontory of the cochlea.
7. The round-window area (any overlying membrane is removed).

The headlight and 12 in. binocular loupe are removed at this time. A Welch-Allen Electric Otoscope is substituted for the actual mobilization procedure. This instrument provides good illumination, adequate magnification, and stability of the speculum for countercontrol of the fingers during the delicate mobilizing movements.

Various Methods of Applying Mobilizing Pressures.—The convex curve of a dulled Turner needle is gently placed on the distal portion of the long process of the incus or the posterior edge of the capitulum of the stapes, depending on the anatomical variations encountered. The incus is not used for mobilizing movements in the following situations:

1. If the incudostapedial joint is too freely movable.
2. If the incus is eccentrically placed on the capitulum of the stapes.
3. If the incus is split or dislocated at the time pressure is applied.
4. If the incus is not at right angles to the underlying crura of the stapes.

Gentle mobilizing movements superiorly-inferiorly and then obliquely are instituted, as already described. As soon as slight motion is felt and observed along the longitudinal axis of the footplate margin, pulsating manual pressure is then applied in direct alignment with the posterior crus. As soon as footplate fracture occurs along its otosclerotic margins or by fracture of the footplate itself, a posterior tilting with a sagging of the stapedius tendon will be felt and observed.

Figure 15

Fig. 14.—Electric otoscope.

At this point the patient often becomes aware of a change in the quality and intensity of his own voice and the voice of the operator. Slight vertigo and a change in tinnitus are also frequently noted.

If the posterior tilt has been rather marked, increased bleeding in the footplate area and occasional perilymph escape may

be noted. This more excessive posterior tilting is usually indicative of an annular ligament tear.

All blood from the middle ear, including the round-window area, is carefully aspirated, and the eardrum and skin flap are replaced. Another otometer reading is obtained at this stage. If a significant improvement on the audiogram is noted, the prognosis is indeed good. If the hearing level is essentially unchanged, or even depressed, it does not necessarily indicate ultimate failure of the procedure. In a number of such cases hearing will improve to serviceability and in some instances the bone-air gap will be entirely eliminated within a month after surgery.

The skin flap is allowed to heal without any packing being applied in the ear canal.

Postoperative Care.—A mastoid head dressing remains on the patient until he is discharged from the hospital the next morning.

Three essential instructions are given to the patient following the stapes mobilization procedure.

1. Avoid blowing the nose.
2. Avoid air travel.
3. Avoid getting water in the ear canal for a period of three weeks.

An oral antibiotic of choice is prescribed at the time the patient is admitted to the hospital and is continued for three postoperative days, as a precautionary measure.

A postoperative audiogram is obtained at three weeks, four months, and one year following surgery.

Surgical Complications

A number of surgical complications can occur during mobilization surgery, as follows:

1. Splitting or Dislocation of the Long Process of the Incus.—Occasionally as the needle is placed on the incus a split in its long axis over the incudostapedial joint or complete dislocation of the incus on the capitulum may occur. In this event one places the mobilizing needle on the capitulum of the stapes for the mobilizing motions.

2. Split Neck of the Stapes.—As pressure is exerted with the needle on the capitulum, the neck of the stapes may split. In this event the incus is separated from the capitulum by an incudostapedial knife and dislocated anteriorly. A capitulum cup is placed over the stapes head and neck, through which the mobilizing pressures are exerted (Figs. 6*A* and 6*B*). This complication with dislocation of the incudostapedial joint does not seem to interfere with the ultimate hearing result, providing the incus is replaced on the capitulum at the time surgery is completed.

3. Crural Fracture.—Crural fracture usually results from too much pressure or from pressure being applied without correct alignment with the posterior crus. If posterior crural fracture occurs, or if fracture seems imminent if further pressure is applied, direct footplate methods are then used, as already described.

4. Excessive Manipulation of the Stapes. If the posterior tilt is excessive, it usually indicates a tearing of the annular ligament.

Although the initial hearing gain may be greater in these cases, I am inclined to believe from my own statistics that these cases are more likely to recede subsequently than are those in which a marginal fracture occurs in the otosclerotic bone without a tear in the annular ligament.

5. Absolute Footplate Rigidity.—In some instances footplates will be encountered which are absolutely rigid and therefore impossible to safely mobilize by any means. In these instances I withdraw and advocate fenestration (if suitable) rather than jeopardize the integrity of the inner-ear mechanism. Shea[12] and Wullstein[12] are suggesting surgical removal of the stapes and footplate and replacement of this bone with a plastic substitution; Rambo[12] suggests surgical removal of the stapes and footplate and placing the eardrum directly over the opened oval window. These suggestions are indeed most interesting.

6. Facial Nerve Injury.—Temporal facial involvement may occasionally occur from the local infiltration of the local anesthetic on the anterior and inferior walls of the ear canal.

Permanent injury of the facial nerve has been known to occur due to instrumental injury of the transverse portion of the facial nerve. Should this accident occur, immediate decompression of the Fallopian canal is indicated.

7. Inner-Ear Complications.—The inner ear may be damaged due to surgical trauma, postoperative hydrops, or postoperative inner-ear infections.

I have observed all three of these complications in my own cases, and in each instance total loss of inner-ear function resulted. Fortunately, these complications seem to occur in less than 0.5% of the cases operated on. Nevertheless, because of this possibility, I rarely operate on both ears simultaneously and likewise operate on the better hearing ear only under most unusual circumstances.

8. Eardrum Perforations. — If marginal perforations occur at the time of surgery, simple approximation will usually result in successful healing. Fortunately, permanent eardrum perforations are indeed rare.

Indications for Stapes Mobilization

1. Every patient suitable for fenestration is also suitable for stapes mobilization.

2. Many patients are suitable for mobilization who would not be eligible for fenestration surgery. Examples include the aged, debilitated, unilateral cases, and patients with rather far advanced cochlear deterioration. A typical example of this type of case is as follows:

A 64-year-old woman with a severe bilateral hearing loss experienced a dramatic restoration of hearing through stapes mobilization. Preoperatively, her hearing loss averaged 75 db. by air conduction and 35 db. by bone conduction in the three speech frequencies. Postoperatively the response by air exceeded the preoperative bone-conduction curve. The three-week hearing test revealed an average of 30 db. by air conduction. This was a surgical gain of 45 db. and restored functional hearing to this severely handicapped person.

A carefully controlled study of bone conduction by me in the operating room immediately before and after mobilization indicated marked shifts sometimes occur. These immediate bone-conduction shifts seem to have little or no relation to the air-conduction changes. The explanation for this phenomenon is not clarified.

3. Any patient with an average difference of 20 db. or more between the bone conduction and air conduction in any three frequencies may be considered suitable for mobilization, regardless of the bone-conduction level.

Results of Mobilization Surgery

To date a total of approximately 800 mobilizations have been performed by me. A detailed statistical report will be published regarding these cases in the near future. A few general conclusions, however, based on the first 400 cases, are of interest at this time.

1. In ideal cases with a bone-conduction loss of 10 db. or less in the three speech frequencies, approximately two-thirds of the patients mobilized by the above methods reached the 30 db. level, or better, by air conduction and maintained their improvement for one year or more.

2. Considering other than the ideal cases, where the bone-conduction curve was depressed below the 10 db. level, the following results may be reported:

(*a*) Approximately one-half of the cases with a bone-conduction loss of 11 to 20 db. at the 500 and 1000 frequencies and 30 db. or less at the 2000 frequency achieved the 30 db. level postoperatively.

(*b*) Approximately one-fourth of the cases with a bone-conduction loss of 20 to 30 db. at the 500 and 1000 frequencies and with a loss of 30 db. or more at the 2000 frequency reached the 30 db. level postoperatively.

3. Reports of postoperative results in terms of achievement to the 30 db. level do

not reflect the degree of hearing improvement for many cases in terms of patient satisfaction. Perhaps a better way of expressing changes in hearing level following mobilization surgery is to consider the amount of closure of the gap between the air-conduction and bone-conduction curves on the audiogram.

Of these first 400 cases the following tabulation of closure of the bone conduction-air conduction audiometric gap is reported as follows:

(1) 67% of the cases operated on closed the bone-air gap 25% or more.

(2) 33% closed the bone-air gap by 50% or more.

(3) 25% closed the bone-air gap by 75% or more.

(4) 11% of the cases operated on closed the bone-air gap completely.

4. Five per cent of the cases operated on were worse by 10 db. or more in the three speech frequencies four months after surgery. Of this 5%, 4% were worse by 10 to 20 db. and 1% of the cases operated on were worse by more than 20 db. in the three speech frequencies.

5. A comparison of the first 400 cases with the last 200 cases operated on reveals an improvement in the over-all results. This improvement may be measured in terms of more cases achieving the 30 db. level, more cases obtaining a greater degree of closure of the bone-air gap, and fewer cases classified as failures. As mentioned previously, a more detailed analysis of results will be published in the near future.

Summary

I routinely advocate stapes mobilization as the surgical approach of choice in patients with clinical otosclerosis.

If stapes mobilization is not successful, subsequent fenestration may be carried out providing the patient is a suitable candidate for fenestration surgery. My experience indicates that a previous mobilization attempt in no way interferes with the subsequent fenestration results. A delay of at least four months is recommended between these procedures.

The surgical approach used in mobilization is of extreme value in exploring the middle-ear area and in treating many types of middle-ear pathology other than clinical otosclerosis.

There are many different techniques being employed at the present time in mobilization surgery. Because of the variation in the anatomy of the stapes crura and footplate, and the variation in the histopathology of otosclerosis, I do not feel any standard routine technique will be developed for this procedure. Each case presents its individual problems, which must, of necessity, tax the ingenuity of the otologic surgeon.

The more experience one has in this type of surgery, the more one realizes that the word "simple" should never be attached to this procedure. Stapes mobilization is a very painstaking surgical procedure, not without the possibility of complications.

The revival of the stapes mobilization procedure by Rosen and the present activities of many competent operators performing this surgery are indeed both stimulating and gratifying. This surgery creates a challenge with new responsibilities and obligations for the otologist.

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Observations of Five Hundred Cases of Transtympanic Mobilization of the Stapes

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The new Rosen[1] technique of mobilization of the stapes in otosclerotic deafness has stimulated the interest of all otologists. At first, the idea of mobilizing a securely fixed stapes and expecting it to stay mobile did not seem possible. Nevertheless, the early results were encouraging because it was clearly demonstrated that normal or near normal hearing could be obtained by reestablishing ossicular conduction. The next step was to do sufficient numbers of cases and wait so that time itself could be the final judge.

My first report on this subject appeared in the ARCHIVES, in May, 1955,[2] at which time 50 consecutive cases were reviewed with audiograms of all those that were successful. The first case was done in March, 1954, two and one-half years ago. Since then, a total of 500 operative cases have been performed and observed, so that now certain conclusions can be fairly definitely established.[3]

The basic trend that was noted in the first 50 cases has carried over with little change through my entire series.

(1) Thirty-six per cent is the over-all number of cases that obtained a Group A improvement in hearing. This includes all cases that were selected for surgery, so that there was a wide variation in the bone-conduction loss. A Group A result is classified as one where the air conduction comes within 20 db. of its bone-conduction potential or where the air conduction reaches the 30 db. level or better in the speech frequencies. This general classification is necessary because cases with varied levels of bone conduction are accepted for surgery. For example, a case with bone conduction at or slightly below the 30 db. level may obtain a postoperative air-conduction level at 35 or 40 db. after a gain of 30 db. This certainly must be classified as a successful mobilization. Each case does not have the same potential to improve, so that all cases that do not reach the 30 db. level cannot be called failures.

(2) There were 176 Group A results obtained in this series of 500 cases. One hundred thirty had air conduction at or above the 30 db. level in the speech frequencies. Forty-six cases obtained Group A results by our bone-conduction potential standard but did not obtain the 30 db. level, because of their poorer bone conduction preoperatively.

(3) Forty-four per cent of the early cases (8 out of a total of 18) where the air-conduction loss did not exceed 35 db. in the speech frequencies obtained Group A hearing.

(4) Beyond the level of 40 db. there is generally much greater stapes fixation, but this certainly is not a fixed rule. There was a sizable group of cases which showed no relationship between the degree of fixation and the level of hearing loss (i. e., a hearing loss at 65 db. may have only a slightly fixed stapes).

(5) When the air-conduction loss drops to the 65 db. level, the stapides are so securely fixed that only one in seven cases can be successfully mobilized. At the 50

Accepted for publication Aug. 8, 1956.

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Prepared for the Northeastern Section of the International College of Surgeons meeting at York Harbor, Maine, July 3, 1956.

db. level one in three can be mobilized, and at the 35 db. level one in two cases can be mobilized. There is no doubt that early cases have a better opportunity of being mobilized, but this is the group that has the greatest percentage of recurrence of stapes fixation.

(6) The physiologic all-or-none law applied. The hearing improvement is either good (considering the original level of bone conduction), or there is none. There are very few in-between results.

(7) The average gain in air conduction in successful cases is seen in Figure 1.

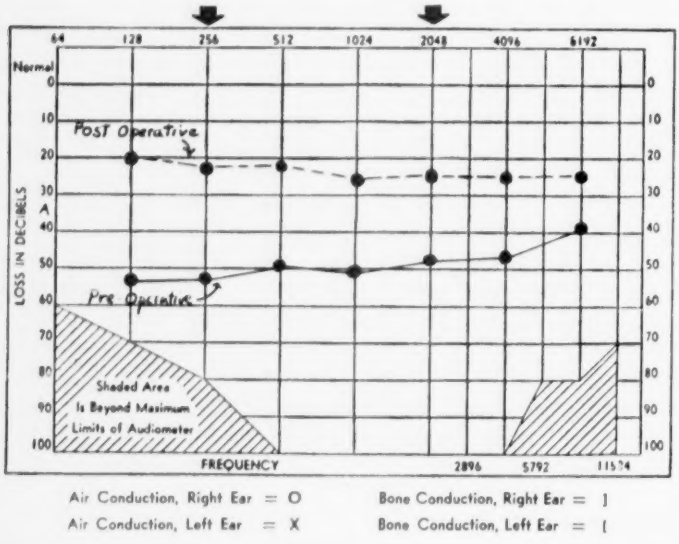

Fig. 1.—Average preoperative and postoperative audiogram.

(8) The tinnitus subsides completely or is greatly diminished if the hearing is improved. There is no change in the tinnitus of unimproved cases.

(9) The degree of stapes fixation can be estimated quickly with the first application of the mobilizer. The stapes that is not securely fixed has a springy sensation when pressure is applied. If the stapes is not mobilized on the first six to eight attempts, the chances of failure greatly increase.

(10) There are two ways in which the stapes may become mobile:

Type A: Gradual mobilization, where the stapes is slowly worked loose. These stapides are less fixed at the start and often when mobilized have the "set-in-rubber" sensation described by Bellucci.[4]

Type B: Sudden mobilization, where the more securely fixed stapes suddenly loosens and becomes extremely mobile.

The hearing obtained in Type B is more permanent than that of Type A, which can be lost in the first 48 hours or first week.

(11) There has been a recurrence of the hearing loss in 35 (20%) patients of the Group A cases. The air conduction in these cases returned approximately to its preoperative level. The majority of these patients lost their initial result within five to six weeks. Several progressed slowly during a period of 10 months. Beyond this period, there seems to be no further change in the initial results.

(12) All cases that were revised (19 patients) were found to have an equal or greater degree of fixation when compared with that at the original mobilization. Eight patients have had a permanent return of a Group A result following a revision. If the revision is successful, the hearing gained has little tendency to deteriorate.

(13) The hearing of many cases may have a "settling down period" within the

first two weeks following surgery (Fig. 2). This is a loss of 5 db. that does occur in several frequencies before the ear is completely healed. It will only be observed if audiograms can be taken starting with the first postoperative day.

(14) Complications have remained at a minimum. There was one case of acute parotitis, probably the result of injecting the local anesthesia. One case of facial paresis appeared three days postoperatively and subsided completely in three weeks. Infections of the middle ear and external canal developed in 25 patients. All subsided with local and systematic therapy. One patient developed an acute labyrinthitis following work at the footplate. It gradually subsided within a period of three weeks, at which time there was further permanent impairment of hearing. Perforation of the tympanic membrane cannot be considered a complication, since, with few exceptions, it will heal quickly. Section of the chorda tympanic nerve will produce distressing symptoms.

(15) There is a broad group of otosclerotic patients that can be helped through the mobilization procedure. However, because there is a great variation in the bone-conduction potential, each patient must be made to understand his own situation. It is the surgeon's obligation to have a frank discussion with each patient, explaining the possibilities that exist and what degree of improvement is or is not possible. It is better to have the patient on the pessimistic side, for disappointments in this sphere for the hard of hearing are difficult to take.

Cases with normal bone conduction may obtain normal or near normal hearing (Fig. 3). Patients with auditory nerve deterioration may obtain as much hearing as their individual bone-conduction potential will allow. Of course, it is desirable to obtain hearing so that it is no longer necessary to use a hearing aid, but this level of hearing cannot be obtained in cases that have poorer bone conduction (Fig. 4). A patient who previous to mobilization could not hear the spoken voice or outside noises without the aid postoperatively may be able to get along with his hearing at close range and be able to hear on the phone. For his work and social relationships he will still have to use his instrument, but with it his hearing will be enhanced, and when he turns it off he

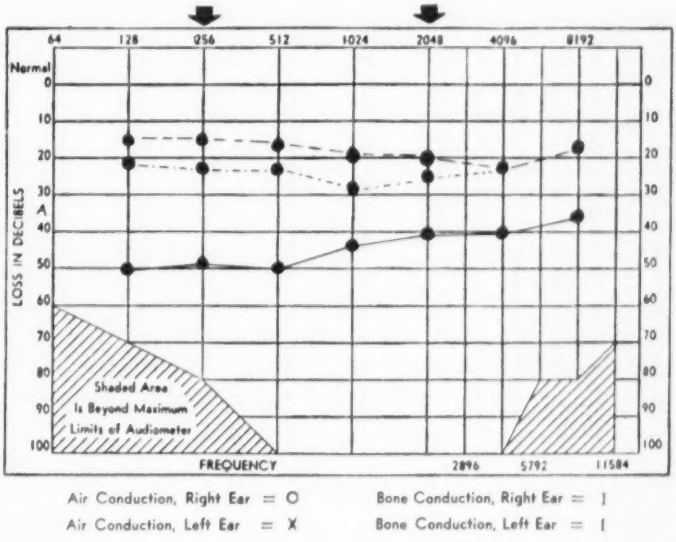

Fig. 2. – Audiogram showing effects of "settling down period." Solid line indicates preoperative level. Dashed line indicates hearing one day postoperatively. Dotted line indicates hearing two weeks later.

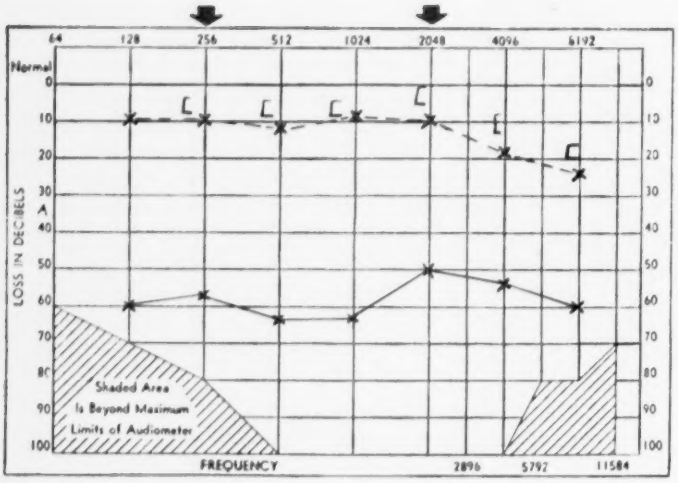

Fig. 3.—Preoperative and postoperative audiogram of patient with normal bone conduction.

is no longer in a vacuum without sound (Fig. 5).

Further Observations with Mobilization Technique

The basic Rosen mobilization procedure is unchanged. A detailed summary of the technique appeared in an earlier article[5] (Figs. 6-12). In the course of doing 500 cases the following additional observations were made.

1. The Use of the "Wabbling" Technique.—The stapes is frequently found to be too firmly fixed to be able to be mobilized with the mobilizer placed on the anterior aspect of its neck. It has been observed that if slight pressure is applied to the inferior

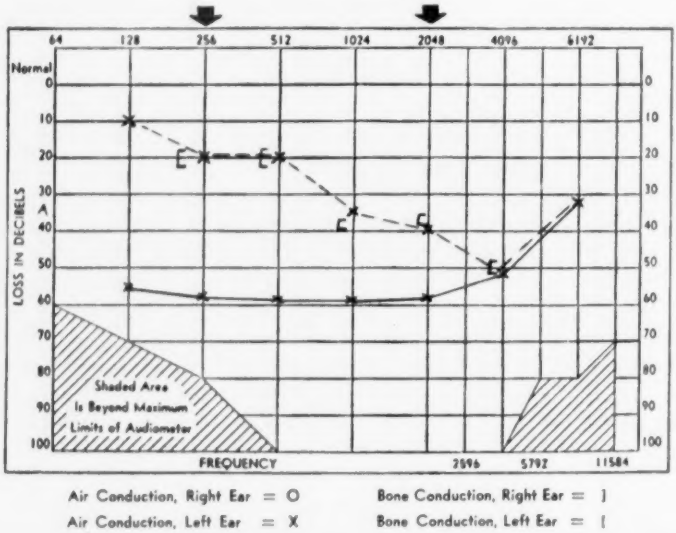

Fig. 4.—Preoperative and postoperative audiogram of patient with a moderate degree of secondary nerve degeneration.

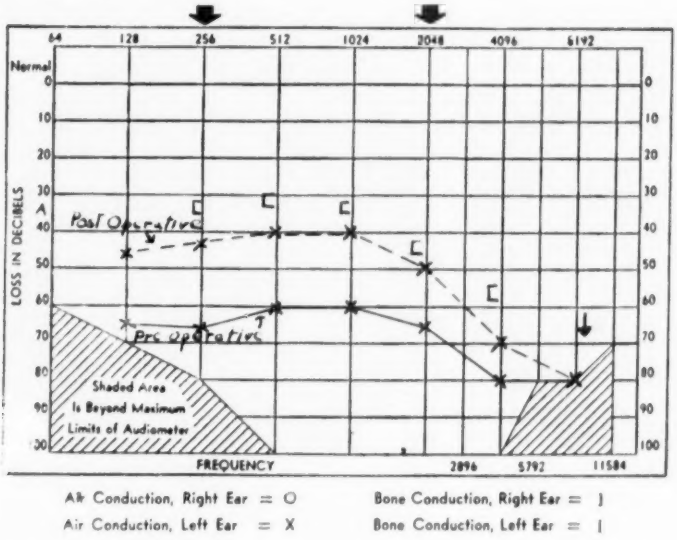

Fig. 5.—Preoperative and postoperative audiogram of patient with advanced secondary nerve degeneration.

aspect of the neck of the stapes, in the direction toward the vertex of the head, the stapes will wabble with a very restricted movement. This small amount of lateral pressure should be a pulsating type and is alternated with a greater force applied to the anterior aspect of the neck of the stapes. Several stapides that would not become mobile did so when the usual pressure (as described by Rosen) was applied only after they had been slightly loosened with the "wabbling" technique. Others, though apparently mobile, did not obtain hearing until greater lateral movement was noted. This wabbling is not used routinely and is suggested only as a means of saving an occasional case that might otherwise be a failure.

2. What Happens to the Hearing if the Incudostapedial Joint Is Opened or the Crura Broken, Provided a Mobile Footplate Has Been Obtained?—Hearing at the 10-15 db. level has been obtained if the stapes has been mobilized and both crura then broken. The same result is obtained if the incudostapedial joint is opened. However, in both instances there must be continuity by contact, either of the broken crura of the stapes or at the opened incudostapedial joint. Once the footplate is mobile, the fractured crura need only be touching to produce a 10-15 db. level of hearing. Should there be any true separation of either the crura or the incudostapedial joint, the air conduction will be at the 50 db. level despite a mobile footplate and good bone conduction. This is in conformity with our concepts of the physiology of hearing. Two mobile windows in

Fig. 6.—Two per cent lidocaine (Xylocaine), adrenalin, hyaluronidase. Complete anesthesia of external canal, drum, and middle ear.

Fig. 7

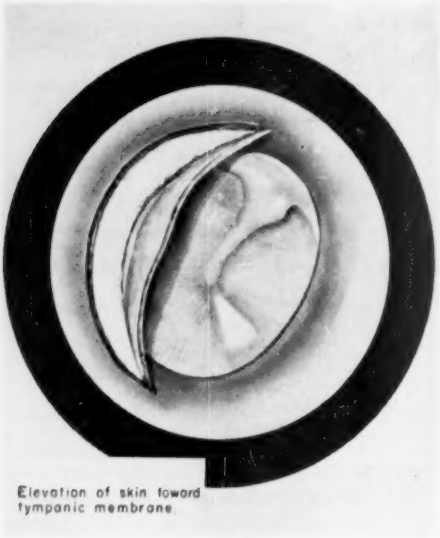

Fig. 8

the middle ear with neither one benefiting from the leverage of the ossicles cannot produce a phase difference in the perilymph, hence there can be no improvement in hearing.

This theory of continuity has its clinical application. An occasional anatomical variation in the position of the incus on the head of the stapes or a narrow external auditory canal may make it technically difficult to apply the mobilizer accurately to the neck of the stapes. The usual application should be tried first, but if the stapes is solidly fixed, the awkward application of the instrument may fracture the crura. Rather than risk a fracture, the incudostapedial joint

Fig. 9

Incus
Stapes
Stapedius tendon
Tympanic membrane folded forward exposing the middle ear. Fixed stapes ready to be mobilized.

Fig. 10

Incus
Alternate appearance of the middle ear where bony annular rim covers ossicles. Dotted lines indicate bony area to be removed.

Fig. 11

can be opened to allow the operator to look directly down on the stapes head. This simplifies the application of the mobilizer on the anterior aspect of the stapes neck before the pressure is applied.

The opening of the incudostapedial joint serves another purpose in these solidly fixed cases that do not yield with the usual technique. The mobilizer can be set directly on the head of the stapes and a rotatory pressure then applied. Should the stapes become mobile, the joint is approximated before the tympanic membrane is replaced. This technique in combination with that used on the anterior neck of the stapes will surely not mobilize all firmly fixed footplates, but it will save the occasional case that might otherwise be a failure.

The direct approach to the head of the stapes can be used with postoperative fenestrations where there has been a closure of the fenestra, instead of proceeding directly to the usual revision. The skin flap and drum are elevated in a similar manner as with the standard mobilization, the incision being modified to conform with the fenestration cavity. This technique will produce useful hearing after the stapes is mobilized if physiological principles are applied. The tympanic membrane has to rest on the head of the stapes to produce a columella effect, otherwise, despite a mobile stapes, the hearing will not improve.

3. Observations Following Perforation of the Footplate.—The observations made at the footplate of the stapes have been interesting and have followed a general pattern. In 35 cases, attempts were made to loosen or perforate the footplate which remained ankylosed after the crura fractured. In all cases, the footplate was directly visualized, since the incus was moved aside and the head and crura of the stapes removed. The size of the "well" in which the footplate sits and the appearance of the footplate itself varied in each case. A thin porous footplate was always associated with a clearly outlined oval window that was not made smaller by the invading pathology. A sharp probe could be passed through this type of plate without much resistance. The denser-appearing footplate was associated with a smaller and often irregular oval window. Firm pressure with a needle proble did not penetrate this type of footplate readily, and if one was not sure where the probe was placed, the impression would be gotten that the pressure was being applied elsewhere and not on the footplate.

Fig. 12

After multiple punctures, the clear perilymph fluid which seeped up always became tinged with blood.

The day following footplate surgery almost half of the patients experienced a marked improvement in hearing, which was first described as hollow or as if heard inside a large echoing chamber. Several of the audiograms taken the first postoperative day had a typical curve (Fig. 13). The low and low middle frequencies reached the 25-30 db. level, only to fall progressively starting at the 1024 frequency. The other type of hearing improvement that resulted from footplate work had a flat audiometric curve at the 25-30 db. level.

It is quite evident that the first week following the surgery is the critical period in so far as maintaining the hearing is concerned. During the first week the hearing of approximately one-half of those patients who had obtained initial improvement returned to or close to the preoperative level. Patients who obtained the type of audiometric curve as indicated in Figure 13 occasionally had an additional loss of 5-10 db. in the upper frequencies.

In this series of 35 patients, 16 obtained a substantial improvement in air conduction but within three to seven days and 8 patients had lost all of the original gain. When the improvement was maintained beyond this period it appeared to be of a more permanent nature. Two patients' hearing became worse following the footplate surgery.

In several cases, the vestibule was opened without any vertigo. Two patients who had very intense vertigo with nausea and vomiting which lasted 6-12 hours had a further permanent loss in air and bone conduction varying from 10-20 db. Those patients whose hearing did improve following perforation of the footplate had a milder degree of vertigo, lasting a few hours postoperatively, followed by several days of unsteadiness.

The hearing gained through this technique will often fluctuate during the postoperative period but never reaches the higher levels obtained through true mobilization of the stapes.

Theoretically, the hearing obtained is unphysiological because there are now two windows exposed within the middle ear without any preferential deliverance of sound. None of these cases have been revised, so that neither of the windows has been reexamined in the successful or unsuccessful cases.

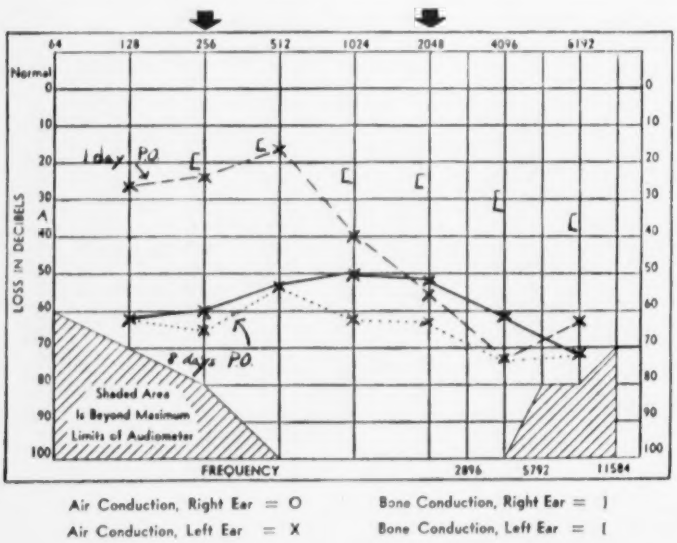

Fig. 13.—Solid line indicates preoperative hearing. Dashed line indicates air conduction two days following multiple perforation of footplate of stapes. Dotted line indicates hearing two weeks later.

Work at the footplate is certainly not without danger to the patient's hearing. Besides the possibility of aggravating the hearing loss, constant disturbing tinnitus may be started as a result of damage to the labyrinth. It cannot be considered a definitive operative procedure that should be done whenever true mobilization fails. Its use should be limited to a few selected cases after careful evaluation. It is not fair to the patient to prevent the possibility of a fenestration being done in the future. Therefore, until a more positive method is introduced, fenestrating or perforating the footplate should be approached with trepidation.

Otosclerotic Deafness

The use of transtympanic mobilization of the stapes as a treatment for otosclerosis has resulted in the restoration of practical and functional hearing in 36% of all otosclerotic patients accepted for surgery. It is quite true that this is not a very impressive figure when compared with fenestration results, but aside from the fact that both procedures can improve hearing, they should not be compared, for they are different in too many respects. Transtympanic mobilization of the stapes cannot and will not ever be the complete treatment of otosclerosis, but it should be assigned its logical place.

Before establishing mobilization's position in the treatment of otosclerosis one must realize that only 36% good results are obtained with the operation. Twenty per cent of these patients will lose their improved hearing as observed thus far. The hearing level obtained with a successful mobilization case is higher than that following the average good fenestration. Accompanying tinnitus will stop entirely or will markedly diminish following successful mobilization.

Mobilization and fenestration must be considered as partners in the surgical treatment of otosclerosis. It should not be a question of competition as to which technique is to be used or preferred. In experienced hands, the mobilization, under local anesthesia, is an innocuous procedure for the patient to go through, and its failure to restore hearing in no way prevents the doing of a successful fenestration two months later.

If the mobilization were a major procedure, like the fenestration, there would be no point in attempting it first when only 36% good results are obtained, but the fact that it is uncomplicated, requiring an overnight stay in the hospital, modifies this thinking.

With patients that are equally suitable for fenestration or mobilization, the surgeon must decide if the patient is the type that would be discouraged after an unsuccessful mobilization and forego a fenestration.[6] If such is the case, a fenestration should be the primary procedure, for percentagewise the patient would then have a better chance to obtain hearing. With other patients, it may be possible to attempt mobilization in each ear, knowing that failure would still not deter them from a fenestration. The surgeon does not have this choice with very early otosclerotic cases or those with advanced secondary cochlea damage, since only mobilization can be advised.

The surgeon who does mobilization must be equipped to proceed with fenestration, since it is his obligation to proceed to the next step in hearing rehabilitation if the patient will allow it.

Summary

The results obtained in 500 cases of transtympanic mobilization of the stapes during the past two and one-half years warrant the continuation of this surgery for the relief of otosclerotic deafness. The percentage of Group A results that can be obtained (36%) was established early in the course of this work. It has not been possible to improve upon this figure using the present operative technique. There is a recurrence of the hearing loss in 20% of cases, approximately half of which are improved when a revision is done.

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Malignant Granuloma of the Face

Contribution to Its Nosology

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Since McBride[18] reported, in 1896, the first case of malignant granuloma, only scattered observations have been published in several countries. Gottesberge,[14] however, presented seven cases. A review of the literature was done recently by Terracol[32] (1954), who called attention to the abundant synonimia, mentioning nasal malignant granuloma, granuloma gangraenescens, sarco lupo pernio, nasal and facial mutilating mortal granuloma, mortal progressive granulomatous ulceration of the nose, gangrenous osteomyelitis of the paranasal sinus, nasal chronic infectious granuloma of unknown cause, idiopathic granuloma of the facial medial tissues, facial mycosis fungoides, pseudotumoral inflammation, malignant reticulosis, malignant mesenchymoma of the nasal-maxillary region, etc. The entity is more frequent in adult males and rare in children.

The most characteristic feature of the disease is the ulcerous-necrotic process, progressing inexorably and resisting any therapeutic measures, as it has been observed in the majority of the cases studied. Its features were described by Stewart[30] (1933), who divided it into the following stages: prodromal, active disease, and terminal stage. The disease starts with an unilateral nasal obstruction accompanied by watery rhinorrhea that becomes bloody and later purulent and malodorous. The rhinoscopic aspect varies, presenting itself as either a purulent rhinitis or a crusty atrophic rhinitis, affecting also the sinus. Ulcerations in the walls of the nasal fossa may be found. There is sometimes a reddish tumor, located either in the inferior meatus or in the nasal septum. The ulcerations spread, destroying the anterior part of the nasal fossa and the surrounding tissues. The osteocartilaginous structure is involved and sequestered. The process may spread to the orbit and to the cribriform plate of the ethmoid. The necrosis then destroys a large part of the nose and of the infraorbital, buccal, zygomatic, and palatine regions invading the pharynx, the esophagus being generally spared; the disease, however, may progress to the hypopharynx, larynx, trachea, and main bronchi. In spite of the wide ulceration the patients present only a slight degree of asthenia, which, except during the final stage, does not affect the vivacity of their behavior. Fever generally occurs in the most varied forms and remains unchanged despite the use of antibiotics. The absence of lymph gland reactions and the lack of metastasis were considered characteristic of the malignant granuloma. It develops by successive fits or with temporary halts. It may last from a few months to 7 years. The blood and cerebrospinal fluid tests do not help in the diagnosis. The bacteriological researches revealed several bacteria, i. e., in order of their frequency: staphylococci, streptococci, fusospirillary associations, diphtherial and pseudodiphtherial bacilli, etc.

Other clinical modalities were catalogued as malignant granuloma because of the sim-

Accepted for publication June 27, 1956.

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ilarity of their clinical behavior to that of the nose. Initial buccal, pharyngeal, and cutaneous forms (nasal wing and sulcus nasolabialis) were thus described (Williams,[35] Pirodda,[23] Vilanova,[34] Alajmo[1]).

The differential diagnosis includes syphilis, tuberculosis, mycosis, leishmaniasis, piantic cancrum, tuberculoid leprosy, atrophic rhinitis, rhinoscleroma, mutilating rhinopharyngitis or gangosa, noma, agranulocytosis, and trophic ulcers.

The treatments that were to a certain degree successful were radiotherapy, hormonal therapy (cortisone and corticotropin [ACTH]), and surgery.

The various names given for the malignant granuloma reflect the uncertainty of its nosology and the variable histological picture of the reported cases. Generally, however, the latter is described as nonspecific. It has been identified as malignant neoplasma, malignant reticulosis, infection by bacteria, spirilla or fungi, and as an allergic process. Recently Brandt[5] considered it moniliasis. Lately, however, the allergic hypothesis and the neoplastic one, specially as tumor of the reticuloendothelial system, have been emphasized.

Report of Cases

Case 1.—A 36-year-old white woman was admitted to the Hospital das Clínicas on April 12, 1955. Her symptoms started 11 months before, when she noted a swelling in the right paranasal region accompanied by stuffiness of the nasal fossa of the same side. Five months later she observed spongy flesh protruding through the right nare, and a malodorous purulent discharge. She had been operated on for both maxillary sinuses, in another hospital, without any improvement. After this operation, the right side of the face, including the eyelids, became intumescent and inflamed. She had noticed three months before a fistula with purulent discharge just below the right inferior eyelid. Air and pus were eliminated through another fistula, that appeared 15 days after the first one. The past history was negative.

Examination.—The physical examination revealed a well-nourished patient. The right paranasal region was increased, congested, and infiltrated. The swelling spread to the nasal pyramid, producing a deviation of the tip to the left side and attaining the right eyelids, keeping them

Fig. 1 (Case 1).—Appearance of patient before treatment.

closed. Two fistulas, partially covered with necrotic tissue, expelling air and malodorous pus, were seen in the regions described above. The anterior rhinoscopy revealed that the external wall of the right nasal fossa was edematous to such a degree that it obstructed the whole cavity. The mucosa of the left nasal fossa was congested. The mouth examination revealed an edema in the right half of the hard palate. The other organs, including the lymphatic ganglions, presented no abnormalities.

X-ray of the paranasal sinuses showed a density shadow of the soft tissues projected on the level of the orbital region and right maxillary sinus.

Hemogram.—White cell count 5000 per cubic millimeter; differential count showed marked deviation to the left, eosinophilia 10%, and relative lymphocytosis 30%; blood serology was normal. The urine was normal. Electrophoresis of the blood serum is shown in the Table. First biopsy presented a nonspecific pseudomembranous inflammation.

A treatment with various antibiotics (penicillin, streptomycin sulfate, oxytetracycline [Terramycin]) was prescribed for 23 days; there was no improvement in the local aspect, and the temperature was not changed. Thereafter cortisone, 50 mg. every 6 hours, was added to the treatment for 21 days. The temperature went down, and there was an evident local improvement.

On April 19, a large surgical excision of the necrotic material was performed as well as the extraction of a sequestrum of the frontal process of the maxilla. Microscopic examination (six blocks): The sections showed a very cellular neoplastic tissue infiltrating the dermis and hypodermis. The surface was necrotic and ulcerated. The cells formed dense or more loose accumulations, with occasional small necrotic areas. In the loose ac-

Case 1: Electrophoresis of the Blood Serum

Date	Relative Per Cent						Absolute Values						
	A	α_1	α_2	β_1	β_2	γ	Total Proteins	A	α_1	α_2	β_1	β_2	γ	Mucoprotein as Mg. of Tyrosine/ 100 Ml.
25/ 4/55	44.3	11.4	12.2	12.7	3.9	15.5	6.44	2.85	0.73	0.77	0.81	0.25	0.99	7.75
3/ 5/55	46.5	5.8	7.4	8.5	6.8	24.9	6.89	3.06	0.38	0.48	0.56	0.44	1.64	6.41
9/ 5/55	40.0	9.5	12.3	6.5	5.7	25.9	7.22	2.89	0.68	0.88	0.47	0.41	1.86	7.87
25/ 5/55	36.2	9.7	9.4	15.2	..	29.5	6.72	2.43	0.65	0.62	1.02		1.97	7.87
25/ 6/55	32.4	7.6	12.1	15.4	..	32.4	6.44	2.08	0.49	0.78	0.99		2.08	7.99
28/12/55	34.3	8.8	16.6	13.0	..	27.3	5.12	1.75	0.45	0.84	0.66		1.39	10.00
Average	38.95	8.80	11.65	11.88 14.02*		25.92		2.51	0.56	0.73	0.75 0.93*		1.65	7.98
Normal Average†	53.14	5.74	9.30	11.17		20.57		3.87	0.41	0.67	0.80		1.49	2.56

* Average obtained by adding β_1 to β_2.
† Normal values of 44 cases from our Laboratory per electroforesis and mucoproteins (Décourt, Ferri, and associates).

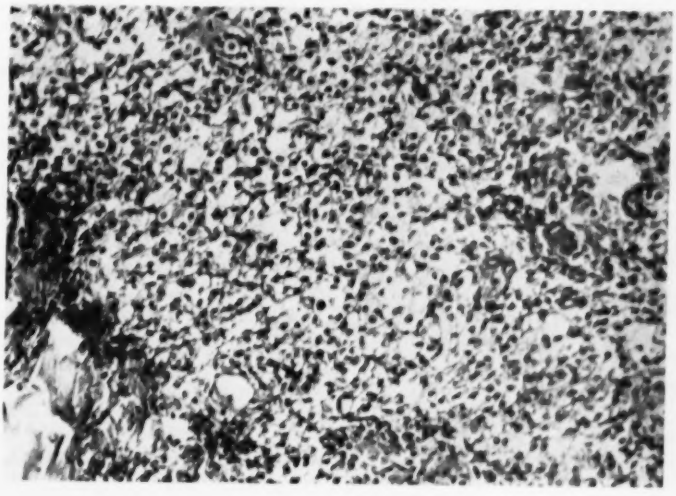

Fig. 2 (Case 1).—Reticulosarcoma: The cellular processes fusing together form a syncytium; hematoxylin and eosin stain; reduced slightly from mag. × 336.

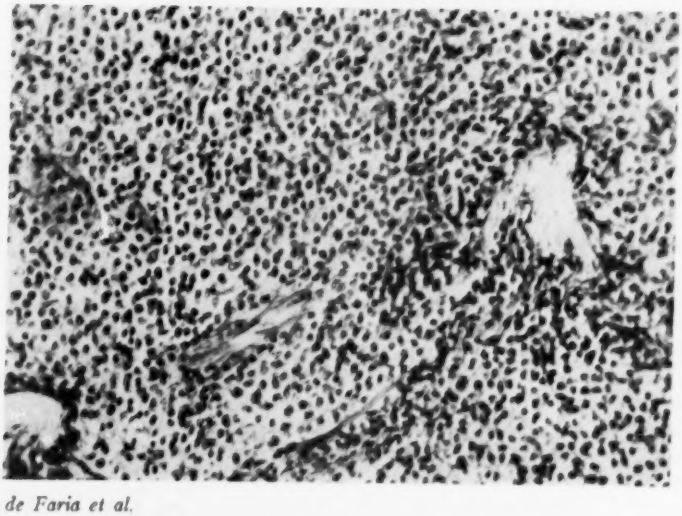

Fig. 3 (Case 1).—Reticulosarcoma: Monocytic-like cells liberated from the syncytium; reduced slightly from mag. × 360.

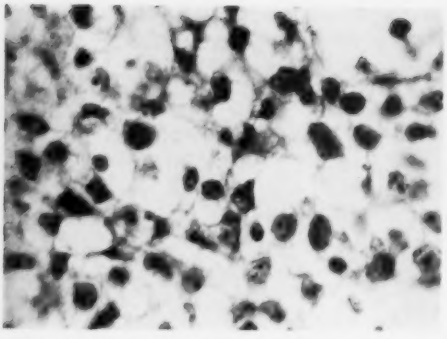

Fig. 4 (Case 1).—Same field as that shown in Figure 2: At left a monocytic-like cell is freeing itself from the syncytium; above this cell a round corpuscle is phagocytized; reduced 2/7 from mag. × 880.

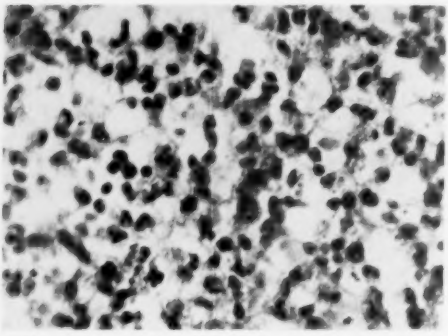

Fig. 5 (Case 1).—Reticulosarcoma: Phagocytic test. Many reticular cells phagocytized the injected iron, which appears as fine granules in their cytoplasm. Perls technique (Prussian blue); reduced 2/7 from mag. × 620.

cumulations they were polyhedral or stellate in shape presenting clear, large, not rarely vacuolated cytoplasm provided with thin or thick processes; these fused with similar ones of adjacent cells, forming a network or syncytium (Fig. 2). The nucleus was clear, vesicular, of round, ovoid, or irregular shape with conspicuous nucleolus. A few areas, showing cells with thin processes, presented the picture of the so-called stellate-cell sarcoma. In the dense accumulations the syncytial pattern was not conspicuous, the cells were more closely packed, with a tendency to assume a rounded form and to liberate themselves from the cellular network; a few free monocytic-like and lymphocytic-like cells as well as rare plasma cells were found in these areas (Fig. 3). Such rounded free cells were scarse in the syncytial areas. However, cells loosening themselves from the cellular network were occasionally seen there (Fig. 4). Mitotic figures were frequent throughout the tumor. No giant cells were seen. Fat staining was negative. The neoplastic tissue had few blood vessels. Reticular fibers formed a conspicuous network in the tumor, which was scarser in the syncytial areas. Near the ulcerated surface there were, between the neoplastic cells, frequent lymphocytes, plasma cells, eosinophils, and mainly neutrophil leucocytes. A few areas at the surface resembled nonspecific granulation tissue due to these inflammatory cells and many newly formed blood vessels. Diagnosis: mixed or polymorphic reticulosarcoma.

In order to verify the phagocytic activity of the tumoral cells a test according to the Senear and Caro technique was performed. A 10% iron saccharate solution was injected into the lesion, and a biopsy was done 48 hours later. This test showed that many of the elements of the cellular network were undifferentiated mesenchymal cells without phagocytic activity, and others, here and there, presented this phenomenon, as revealed by the Prussian blue technique (Fig. 5).

During the phagocytic test the cortisone treatment was interrupted; an aggravation of the lesion and of the patient's general condition was noticed. With resumption of cortisone therapy there was once more an improvement. A radiotherapeutic treatment of a total of 4,800 r was begun on June 13, 1955. On July 13, 1955, the patient was discharged because of the complete cicatrization of the lesion and temperature normalization. Partial facial destruction was the remaining sequela. On Dec. 26, 1955, she was readmitted because she had become feverish again, had pains all over her body, was constipated, inappetent, and vomited. It was not possible to elucidate the cause of these disturbances. But the facial lesion was quiescent. No lymphoid ganglions were felt in the neck. She died on Jan. 17, 1956.

Necropsy.—At the face wide destructive process, involving the right lateral surface of the nose and the adjacent infraorbital region to the orbital floor, destroying the soft tissues, the nasal bone, and part of the maxillary bone. The neighboring skin was noninfiltrated. No cachexia. Slight icterus (skin, conjunctivas, intima of the aorta). Bronchopneumonia in both lungs (a small focus in the left apex and a large one in the upper right lobe), with serous pleuritis at right (about 400 ml. of fluid). The heart (200 gm. weight) presented a neoplastic flattened nodule (5 cm. high and 8.5 cm. wide), involving the basis of the left ventricle, from the anterior to the posterior surfaces. The neoplastic tissue was white-yellow, compact, and infiltrated in this area the myocardium, which was there 1.5 cm. thick (the noninfiltrated myocardium of the left ventricle was 0.9 cm. thick and that of the right one 0.3 cm.). Liver enlarged, yellow, soft, weighing 220 gm. Gallbladder and extrahepatic biliary ducts without changes. Spleen

moderately enlarged (380 gm.), hyperemic, also soft; Malpighian bodies not conspicuous. Kidneys without sharp distinction between the cortex and the medulla, weighing (right) 200 gm. and (left) 160 gm.; voluminous calculus molding the left pelvis and calyxes with amorphous internal structure (phosphate stone). Red-yellow nodule, without sharp limits, about 3 cm. in diameter, in the myometrium of corpus uteri. The lymph nodes were small, but one in the right submandibular region and another in the lumbar region were enlarged. The former was larger (1.5 cm. in the greater diameter) and soft, the parenchyma being substituted by a pink-colored tissue. The other node showed only a small area (2 mm. in diameter) of similar tissue. Bone marrow of the sternum and of the bodies of the lumbar vertebrae without changes.

Microscopic Examination.—Skin and deep tissues delineating the facial ulceration (three blocks) showed an infiltrating neoplastic tissue composed of free polymorphic cells, of various sizes. Generally, however, they were rounded monocytic-like elements with voluminous nucleus, conspicuous nucleoli, and scarce cytoplasm. Reticular fibers in moderate number. In the margin of the ulceration there was copious cicatricial tissue infiltrated by the neoplastic cells. The striated musculature was also infiltrated by the latter. The right submandibular lymph node presented a similar neoplastic tissue, substituting the most part of the normal structures and infiltrating, in an area, the capsule as well as the adjacent tissues. However, between the isolated cells there were many syncytium-forming ones. The nodule in the heart showed the same picture as the facial neoplastic tissue, but with frequent elongated and oval cells.

Liver: Besides intense fat metamorphosis of the liver cells and necrosis of single ones in the central portion of the lobules, there were conspicuous enlargement, multiplication, and liberation of the lining cells of the hepatic sinusoids. These cells were isolated or in groups of three to four elements. They presented the feature of malignant cells as described in the skin with larger and giant elements (Fig. 6). The latter showed bizarre forms and two to four nuclei, and occasionally more. The nuclei were polymorphic and frequently hyperchromatic. Small foci of neoplastic cells were found at the periphery of lobules around the interlobular spaces.

Spleen: Picture of acute splenitis. Throughout the red pulp and rarely in the white large isolated pleomorphic cells with malignant feature, as seen in the liver. Occasional giant cells showed phagocytosis of red blood cells.

Uterus: Infiltrating neoplastic tissue of the reticulosarcomatous type (see skin), with uncommon giant cells. In the renal cortex a few small foci of neoplastic cells with conspicuous, free, large reticular cells, lymphocytic- and monocytic-like elements; giant cells as seen in the liver.

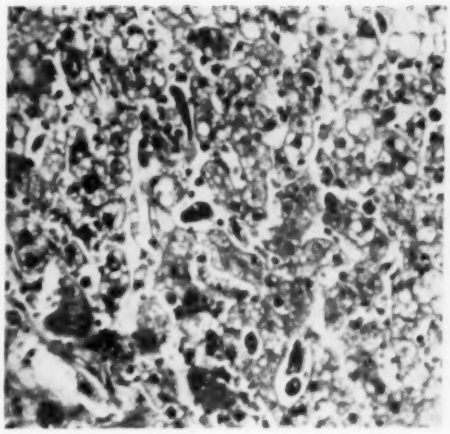

Fig. 6 (Case 1).—Liver: Besides fat metamorphosis of hepatic cells note the polymorphous voluminous neoplastic cells in the widened hepatic sinusoids; hematoxylin and eosin stain; reduced slightly from mag. × 360.

Diagnosis: Facial reticulosarcoma; metastasis in the right submandibular lymph node, heart, and uterus as well as in the liver and kidneys.

This case led us to a histopathological review of the first two cases of malignant granuloma previously reported by Corrêa and Elisabetsy.[9]

CASE 2.—A 17-year-old boy with medial facial necrosis dying after a seven-month evolution of the disease. Two biopsies of the superficial tissues only were performed, one showing a chronic nonspecific inflammatory process and the other a fibrinous-purulent inflammation. The necropsy showed a wide necrotic process involving the left side of the face, with destruction of the soft tissues and bones of the nasal, orbital, maxillary, palatine, ethmoidal, and sphenoidal regions. In the vocal folds, mainly at the right, a ulcerative process covered by white-yellow exudate. Numerous isolated and confluent bronchopneumonic foci in both lungs. A bronchial node presented caseation. No metastasis was found. Microscopically sections of the skin delineating the facial ulceration showed a neoplastic tissue as described in Case 1, in which predominated the free monocytic-like cells. Mitotic figures were very frequent. The deep tissues, the striated musculature inclusive, were infiltrated by the neoplastic cells. The vocal folds presented an acute necrotizing pseudomembranous process with many bacteria at the surface. Neoplastic tissue failed there.

Fig. 7 (Case 2).—Tissue from the margin of the facial ulceration: Reticulosarcoma infiltrating the striated muscle; hematoxylin and eosin stain; reduced slightly from mag. × 288.

Case 3.—A 27-year-old woman with facial and pharyngeal necrosis dying of the disease. Repeated biopsies did not reveal the diagnosis. However, a review of the slides showed the true nature of the process. At the surface necrotic tissue with heavy infiltration by leucocytes; then a diffuse infiltration of reticular cells forming a syncytium as described in Case 1; frequent hyperchromatic nuclei and a few mitosis. In some areas the presence of many inflammatory cells (lymphocytes, plasma cells, and neutrophil leucocytes) masked the neoplastic ones.

Comment

These three cases of facial malignant granuloma showed a histological picture of reticulosarcoma. It is a question to know whether every case of this clinical entity is of neoplastic nature. Such a question cannot be answered through the analysis of all reported cases. However, there are in the literature many facts supporting the neoplastic nature.

Pirodda and Guenzi,[23] reviewing the histological picture of several malignant granuloma cases published, especially in the German literature, were able to establish the diagnosis of malignant tumor of the reticuloendothelial system in many cases. Later other cases were also diagnosed as of neoplastic nature (Terracol,[32] Bouchet [4]).

The improvement or cure with radiotherapy gives support to the neoplastic nature. Terracol [32] collected eight cases from the literature; six were cured and two improved. To these we can add Vilanova's [34] case, with transient improvement, and Causse's [8] case, that had its fatal evolution arrested by the action of cancerocidal actinomycin and radiotherapy. Good results were also pointed out by Ellis [13] in two cases, Howell [16] in one case, and by Alajmo [1] in one case.

Possibly other reported cases could be also of neoplastic nature, but the histopathological descriptions are insufficient to establish the diagnosis through the critical analysis. Really two of our three cases were reported by Corrêa and Elisabetsy [9] as not being neoplastic in origin. However, in the review of the slides of these cases neoplastic tissue could be detected. The true diagnosis may be difficult for the pathologist examining small pieces of ulcerated tissues, in which many inflammatory elements appear, masking the neoplastic ones. The neoplastic tissue itself may present a polymorphic structure with inflammatory-like cells (de Faria [11]). In one of our cases primitive reticular cells (or undifferentiated mesenchymal cells) and phagocytic reticular cells (Maximow [17]) were found through the vital staining (Fig. 5). Lymphocytic- and monocytic-like cells were also conspicuous in this

tumor. Whether the lymphocytes and plasma cells found in the latter are derived from the neoplastic reticular cells cannot be determined. However, there was evidence that round cells were formed locally (Fig. 4).

The matrix cell in this case is the undifferentiated mesenchymal cell (Maximow [17]), or the dedifferentiated macrophagic cell with a return of the developmental potentialities of the former (Puhr,[24] Businco [7]). The first case may be called polymorphic or mixed reticulosarcoma (Robb-Smith,[25] Rotter and Büngeler [27]) or reticulohistiocytosarcoma (Businco,[7] de Faria [11]) and the other two cases reticulosarcoma. The origin of cells in the reticuloendothelial system is indicated through the phagocytic activity of a few cells and the conspicuous transformation of the Kupffer cells of the liver in neoplastic ones (Fig. 6). Lately other cases of facial malignant granuloma have also been included in the malignant processes of the reticuloendothelial system (Piquet,[22] Terracol,[32] Pirodda and Guenzi [23]).

One of our three cases improved with corticotropin and cortisone. A similar effect was reported by others (Williams,[35] Moore,[20] Hagens [15]). This improvement has been adduced as supporting the allergic nature of the disease. However, such good results with these drugs were not confirmed by others (Breckenridge,[6] McCallum,[19] Rossert,[26] Bandler,[3] Alexander,[2] Sneddon,[29] Sweet [31]). Moore's case, according to his personal communication to Alexander,[2] ended in death. Then the action of corticotropin and cortisone may be on the associated infection, as it was in our case, whose neoplastic process was resistant to this treatment.

The electrophoresis showed in one of our cases that the gamma globulin was kept above the normal level, making us believe there was no defense deficiency, contrary to the opinion of Woodburn and Harris.[37] The mucoprotein always was present in high levels, as reported by Winzler,[36] in malignant neoplasms.

Summary

A case of facial malignant granuloma is described. Only after a large biopsy specimen was taken could the reticulosarcoma diagnosis be determined. In two other cases, previously reported, which had not been initially identified as neoplastic process, a histopathological review showed that both cases were reticulosarcomatous in origin. Using as a basis these three cases and data from the literature, we admit that most of the cases of facial malignant granuloma are of neoplastic nature. The difficulty in establishing the histopathological diagnosis in small pieces of tissue is emphasized.

Rua Jeronimo da Viega, 457 (Dr. Cutin).

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Glomus Jugulare Tumors

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Although glomus jugulare tumor is not very common, it is still sufficiently prevalent not to be considered among those that are rarely encountered. As additional attention is given to this disease, more cases will be uncovered. This is evidenced by the marked increase in the number of cases reported ever since the first report made by Rosenwasser, in 1945,[1] including the very early cases as shown by the presence of an intact drum, reported by Davol[10] and Strong.[12]

Definition

Glomus jugulare tumor includes any tumor that affects the glomus bodies which may be found in the following locations:

1. Adventitia of the dome of the jugular bulb
2. Along the course of the tympanic branch of the glossopharyngeal nerve
3. Along the course of, never over, the cochlear promontory
4. Along the course of the auricular branch of the vagus nerve (Arnold's nerve) as far distal as that point where it crosses the descending part of the facial nerve

Synonyms

The names of tumors which are often used synonymously are as follows:

1. Carotid body tumor
2. Tympanic body tumor
3. Chemodectoma
4. Nonchromaffin paraganglioma system, which includes
   - *a.* Glomus caroticum, or carotid body
   - *b.* Glomus jugulare and paraganglion tympanicum
   - *c.* Aortic-arch bodies, or glomus aorticum
   - *d.* Paraganglion intravagale and juxtavagale, which are closely associated with the vagus nerve

A diagnosis of nonchromaffin paraganglioma is suggested, according to Flynn Jr. et al.,[3] by the following features:

1. Development is in close association with the cranial nerves and their ganglia and the vessels of the branchial arches.
2. Histologically similar, if not identical,[2] and consist of nests of epithelioid cells in a highly vascular stroma.
3. Have an afferent sensory type of innervation from the fibers probably parasympathetic to the different cranial nerves.
4. No chromaffin substance, nor do they secrete epinephrine or any detectable hormones.
5. Function is that of chemoreceptors—able to detect changes in pH or carbon-dioxide concentration in the blood which circulates through the highly vascular network.
6. Tumors arising from them exhibit similar microscopic structure and clinical behavior and seem to have a definite tendency to multicentricity.

Clinical Features

This type of tumor may affect persons of any age, the youngest reported being 17 years old[4] and the oldest 72 years.[6] Both sexes are affected, although it is predominant in females, with a ratio of about 4:1. Most of the symptoms are nonspecific.

1. Mass in the ear—polypoid in character and varies in size. It is hard to differentiate glomus jugulare tumor from the common

Accepted for publication Aug. 10, 1956.

Read at the New Jersey Academy of Medicine (Section of Otorhino-Laryngology), Dec. 12, 1955.

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Awarded Third Prize, Harrison S. Martland Award, sponsored by the Essex County Pathological and Anatomical Society.

aural polyp except for its tendency to bleed [2] and the pulsations which may be elicited by the "pulsation sign test" of Brown.[6] It may also be differentiated by pressure exerted on the carotid artery which may produce blanching of the tumor and cessation of the pulsations.

2. Discharge from the ear—may be thick, purulent, and malodorous like other cases of otitis media.

3. Tinnitus—may be the first complaint offered by the patient and varies in character from mere ticking or "flowing steam" or "roaring noises" in the ears. Usually it is synchronous with the heart beats.

4. Impairment of hearing—varies from very slight loss to total deafness. Audiogram is usually that of conductive type.

5. Pain in the ear—usually is deep-seated. It may also be the first complaint of the patient, and is usually referred to as headaches on the side of the tumor.

6. Facial paralysis—varies in onset according to the location of the tumor in relation to the facial nerve. Those tumors near the facial nerve will have earlier onset of paralysis than those farther from it.

7. Hemorrhage—may be the alarming symptom that brings the patient to the physician. This may be spontaneous or caused by slight instrumentation. Commonly, this is encountered in the clinic, where the otologist may try to remove the polyp as a clinic procedure or in performing biopsy. It could be controlled by packings.

8. Otoscopic findings—in the early cases, there may be a sharply defined reddened or bluish mass in the tympanic cavity behind the intact drum which may be misinterpreted as Schwartze's sign for vascular otosclerotic focus.

9. X-rays—may or may not be an aid to the diagnosis. In the earliest stages, there is no change noted. As the disease progresses, the cells become cloudy and may resemble that of acute mastoiditis before any bony destruction. By the time there is loss of function of the seventh and eighth cranial nerves on the affected side, there may be demineralization which appears like dissolution. A characteristic x-ray finding, if there is any, is the picture of the mastoid and petrous cells to be "rubbed into coalescence."

10. Biopsy—gives the positive diagnostic evidence. The tumor is seen as groups of tumor cells which are bordered by a multitude of thin blood vessels with a single epithelial lining of the capillary or precapillary caliber and surrounded by a reticular network. These groups of cells may form alveolar structures which may not always be distinct and may form compressed, cord-like structures. The cells are epithelioid, large, and polyhedral. Their nuclei are small and hyperchromatic. Cytoplasm is abundant and is frequently vacuolated, which may obscure the cell outlines in some instances. Mitosis has never been reported.

Rosenwasser points out in his article [2] the resemblance of the histologic picture of this type of tumor to that of carotid body tumors.

Biopsy is done by the Rosen technique,[9] wherein incision is made from 3-9 o'clock about 5-7 mm. external to the drum. Then the drum is lifted up out of its sulcus and reflected upward, exposing the lower half of the tympanic cavity. A small piece of tissue is removed from the suspected tumor.

In the late stages of the disease when the tumor protrudes through the external canal outside to the meatus, a small piece of this tissue is removed. However, when doing biopsies, bear in mind the tendency of this tumor to bleed. The patients may lose a lot of blood.

11. Metastasis—has been reported but occurs usually in the late stages of the disease. Metastasis to a cervical lymph node was reported by Winship, Klopp, and Jenkins [13]; to the liver by Lattes and Waltner,[5] and to the liver and lungs by Tamari et al.[14]

Diagnosis

Diagnosis is based on the clinical history and the histological examination. The following are suggestive features [3,7] which should make one suspect the presence of the tumor:

1. Tendency to bleed by the polyp following slight trauma.

2. Sensation of pounding noise and pulsations in the ear, sometimes synchronous with the arterial pulse.

3. Progressive, expansive type of growth which leads to destruction of temporal bone by pressure atrophy in advance cases.

4. Possibility of distant metastasis.

5. Generally long duration of symptoms associated with recurrences of the polypi.

Treatment

Treatment may be either by (1) surgery, (2) irradiation, or (3) combination of surgery and irradiation.

Surgical Treatment.—G. Shambaugh Jr.[7] in his article discussed the surgical approach to this type of tumors. Radical mastoidectomy is performed, and precautions against inciting profuse hemorrhage is undertaken by removing all the bone surrounding the tumor before the actual separation of the tumor itself. Weille and Lane Jr. have written an article[8] about the surgical problems involved in the removal of these glomus tumors. Among them are hemorrhage, possibility of recurrence and possible invasion of the neighboring structures, metastasis, and facial paralysis with the inability to use the conventional anatomical landmarks in the operation.

1. Hemorrhage: Because of the very nature of the tumor, bleeding is commonly encountered in the treatment. Provisions for blood transfusions must be available before contemplating surgery. The hemorrhage could be controlled by packings, and the tendency to bleed is overcomed by the limitation of trauma directly to the tumor at the shortest possible time. This is done by the removal of the bone surrounding the tumor before excising the tumor itself.

2. Recurrence always is possible with or without invasion of the neighboring structures. Because of the slow-growing nature of the tumor and the apparent state of benignancy, a small piece of tumor may be left, especially when it is at the jugular bulb, where there is danger of massive hemorrhage, which may result in shock and subsequent death.

3. Metastases: There are only three cases so far reported in the literature that had metastasis—one to the liver,[5] to the cervical node,[13] and to the lungs and liver.[14] With this as a basis, the danger of excising the origin of the tumor, especially in the jugular bulb, is infinitely a greater risk than accepting the possibility of metastases.

4. Facial nerve paralysis is very frequent and may be due to the surgical difficulty encountered secondary to the profuse bleeding, relation of the tumor to the facial nerve, and inability to use the conventional anatomical landmarks in the operation.

5. Inability to employ the conventional landmarks accounts for the occasional facial paralysis.

Irradiation.—Ever since the very first time this tumor was known, irradiation has been done. This is given empirically. Graf reported eight cases who were treated with deep x-ray and obtained favorable results. Recently, Williams et al.[11] reported 13 cases wherein 1200-2400 r were given within one to two weeks. They have concluded, "We believe that it is fair to conclude that radiation therapy has a definite therapeutic effect on chemodectomas of the glomus jugulare. The results seem to indicate that if surgical procedures are used, they should always be combined with radiation therapy."

Surgical Removal Followed by Irradiation.—Rosenwasser states that irradiation alone, even as administered today, unaccompanied by surgery, offers little encouragement in these cases. Irradiation is being given in spite of the fact that the tumor is known to be radioresistant because of it being able to shrink the tumor. Irradiation is started usually one week after the operation. Doses vary from 1200-2400 r. Others use the tumor dose used to control malignant tumors of the middle ear and mastoid, which is 5000-7000 r. Response of these tumors to irradiation is said to be slow, and the tumor may continue to shrink for several months after the treatment. Ir-

radiation is especially indicated when there is doubt as to the possibility of incomplete removal of the tumor or when part of the tumor is left purposely to avoid profuse hemorrhage.

Report of Cases

Case 1.—A 52-year-old white woman admitted for pounding, throbbing, and roaring sensation with impairment of hearing in the right ear. Examinations on admission showed everything to be negative except in the right ear. Canal was slightly red, but there was no discharge. Drum was dark red and bulging, with the short process of the malleus hardly seen. Umbo was depressed and no light reflex. In front of the short process was a deep depression which was gray in color. Bone conduction was better than air conduction; Weber was lateralized toward the right side. There was no nystagmus. Corneal reflex present and equal on both sides. Past-pointing test was normal.

Preoperative diagnosis: carotid body tumor (nonchromaffin paraganglioma), right.

Radical mastoidectomy was done. Operative findings were nests of vessel formation found lining the few large antral cells. Mass of firm, reddish, vascular-looking tissue appeared to arise from the medial wall of the middle ear extending downward around the Eustachian tube and forward, lifting the external auditory canal and drum. Mass was apparently adherent to the point just medial to the facial nerve directed toward but not communicating with the jugular bulb.

Pathological report: "Glomus Jugulare Tumor."

Case 2.—A 59-year-old Negro woman was first seen in the clinic in 1935 because of pain in the left ear with "bloody discharge." Examinations at that time showed only slight impairment of hearing on that side. Two years later, she was found to have "polyps" in the external auditory canal, which were removed in the clinic, followed by "profuse bleeding." Because of the persistence of the discharge, x-rays of the mastoid were requested and showed "clouding of the cells but walls were unbroken." There was also recurrence of the polyps, and radical mastoidectomy was done with the excision of the "polyps." It was noted on the operating table that there was profuse bleeding coming from the region of the Eustachian tube, which prevented the surgeon from curetting this region.

Pathological report: "granulation tissue" (done in 1937 before the first report of the tumor in 1945).

Patient came back and forth to the clinic because of the recurrence of the discharge and the polyps in the same ear. In October, 1955, a globular mass, purplish in color, cystic in consistency, was noted occupying and obstructing the whole external auditory meatus. This was accompanied by purulent discharge and slight paresis of the left side of the face. There was complete loss of hearing at this time. She was operated on and was found to have a tumor mass occupying the whole mastoid cavity (operative defect due to previous operation) and the middle ear aside from the external auditory canal. There was noted during the operation profuse bleeding, which was thought to be the jugular bulb itself. After the removal of the tumor, the cavity was packed tightly with oxidized cellulose (Oxycel) and the bleeding was controlled.

Pathological report: "Glomus Jugulare Tumor."

Case 3.—A 47-year-old white woman admitted for pains in the right ear that occurred intermittently for several years. Ear had been discharging a foul-smelling purulent material one month before admission. Aural polypectomy was done and specimen sent to the laboratory for histological examinations.

Clinical diagnosis: polyp, aural (right).

Pathological diagnosis: "Glomus Jugulare Tumor," right.

Comment

Three cases are presented with the final diagnosis of glomus jugulare tumor, as confirmed by the pathological report. Case 1 was diagnosed with an intact drum just because the otologist was alert enough and entertained the possibility of the glomus jugulare tumor. Case 2 shows the typical history of repeated, recurrent polyps, with a tendency to bleed, loss of hearing, facial pareses, and the long duration of the disease (20 years), followed by facial paralysis after removal. Review of the slides taken in 1937 showed the presence of the same pathology noted recently. Because of the fact that it was only in 1945 when the first article about such tumors was published, it was not diagnosed then. This should be true in most hospitals. Case 3 shows the importance of sending all specimens obtained in the operating room for histologic examinations. The polyp removed was innocent-looking and assumed in all respects the appearance of a benign aural polyp. Yet histologically, it turned out to be glomus jugulare tumor.

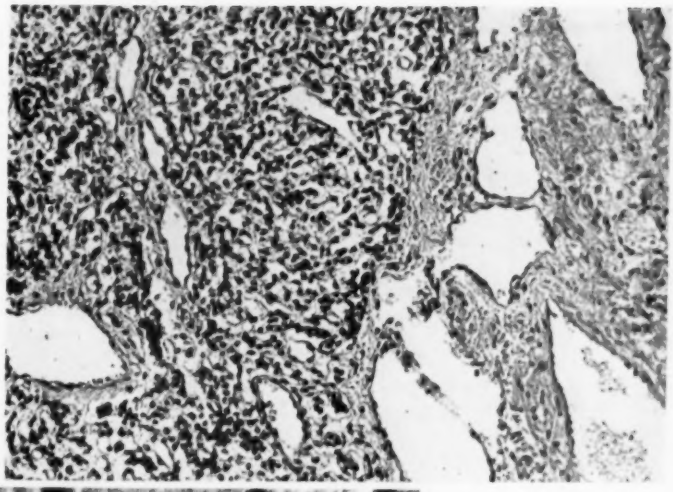

Fig. 1.—Low power shows the groups of tumor cells bordered by thin blood vessels and surrounded by reticular network.

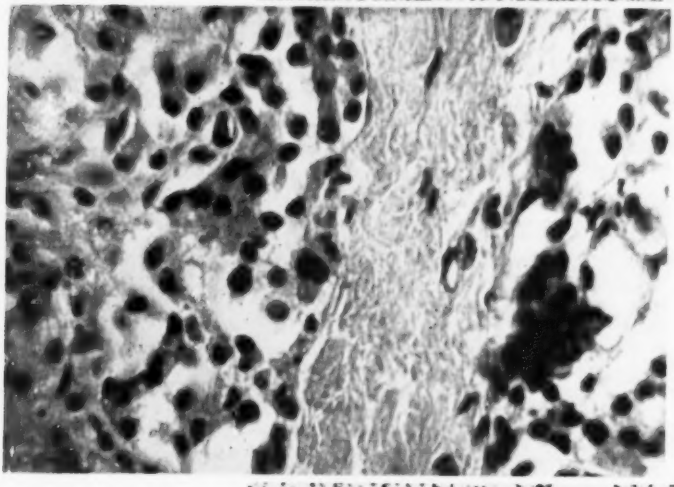

Fig. 2.—High power shows the typical epithelioid cells, which are polyhedral and large. Nuclei are seen to be small and hyperchromatic. Cytoplasm is abundant and often vacuolated.

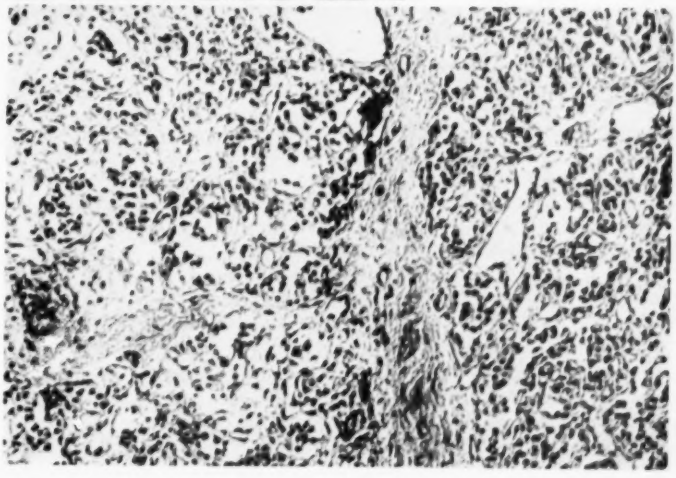

Fig. 3.—The groups of cells as shown in Figure 1. This picture was taken from a slide prepared in 1937 and previously diagnosed as "granulation tissue" (before the first publication by Rossenwasser about glomus jugulare tumors, which appeared in 1945).

The attending surgeons of the Newark Eye and Ear Infirmary granted me the privilege of using their cases. The Pathology Department of the Martland Medical Center took the pictures.

77 Central Ave. (2).

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Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

The Use and Abuse of Hearing Aids

A Study on Identical Twins

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It has been our experience that most patients who wear hearing aids have worn them for some time before consulting us. Occasionally we find that the aid the patient is wearing is not satisfactory and recommend that certain changes be made. They return to their hearing aid dealer to have the changes made, or perhaps to buy a new aid, and usually will not follow our advice to return for retesting to see whether the changes have resulted in any improvement.

In Denver we are fortunate to have the Denver Hearing Society, which is sponsored by the Community Chest. It has a full-time audiologist who gives individual and group instruction in all phases of auditory training and rehabilitation. Even though the charges are minimal ($2.00 an hour for individual instruction and 50 cents an hour for group instruction), most of our patients wearing aids will not follow our recommendation to avail themselves of this opportunity to improve their listening ability. For the patient buying an aid for the first time this training is even more important, but, unfortunately, here too, our patients fail to take the training. As yet, except for the cases reported here, we have been unable to obtain any clinical evidence of the value of such auditory training and rehabilitation.

Guilford and Haug [1] state, "The benefits of educational counsel, with reference to auditory training and lipreading, apply in like degree to those patients with borderline impairments, not requiring a hearing aid, and to those for whom no benefit is possible from amplification." They also report that a patient with nerve deafness improved her discrimination ability from 58% to 89% with 20 hours of formal auditory training designed to make her a more critical listener. They conclude in part as follows, ". . . we feel that the otologist must take the responsibility of reducing the abuses and mistakes in its [the hearing aid's] use. No group of handicapped persons is more open to exploitation by commercial interests than the hard of hearing. This is due in part to the fact that the fitting of no other prosthetic device receives as little professional supervision as does the hearing aid, and nowhere is there as little interest in establishing and maintaining standards of competency for the prosthesis fitter as is found with respect to hearing aid dealers."

The results of our testing on identical twin sisters with similar hearing losses, environment, and auditory training show very vividly the need for supervision in the correct fitting of hearing aids and the advantages obtained from auditory training.

The twins were referred for tonsillectomy and adenoidectomy in November of 1952, when they were 5 years old. Neither one had had any earaches or otitis media, but both had had more sore throats and tonsillitis than normal. They had both had mumps a year previously. Both had learned to talk at the normal age, and their speech was not that of a deafened person. The parents had never suspected any loss of hearing in either twin until September of 1952, when they thought Twin A was not hearing well in the right ear. Unfortunately, only Twin A's hearing was tested on that

Accepted for publication Sept. 3, 1956.

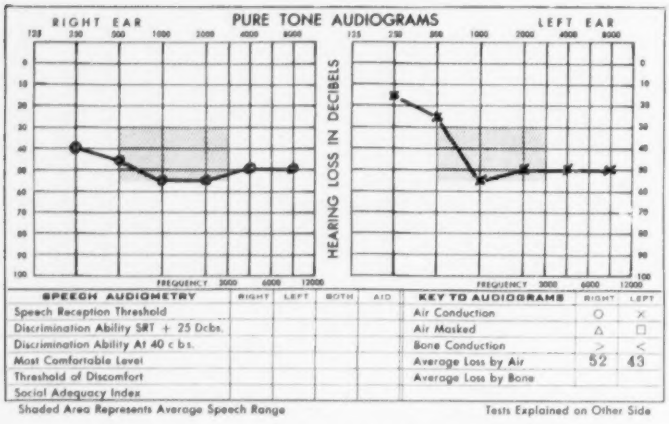


Fig. 1.—Audiograms of Twin A taken on Nov. 10, 1952.

first visit. They both had tonsillectomy and adenoidectomy soon thereafter.

Twin A's first hearing test in November of 1952 is shown in Figure 1. It seemed to be an accurate test for her age and is not much different than later tests. Therefore it is hard to understand why the parents had thought the deafness was only in the right ear. Perhaps the later history of the onset of Twin B's hearing loss is also incorrect.

Twin A was next seen in March of 1953 and her mother thought she heard much better. This was apparently confirmed by audiograms taken at the Denver University Speech and Hearing Center which showed improvement in the left ear, as shown in Figure 2. However, the speech tests would indicate that either the pure-tone threshold or the speech tests were incorrect.

In November of 1953 the mother brought the twins in for retesting because as part of the public school hearing testing program a report was sent home that Twin B also had a hearing loss. The parents had not been aware of any loss and were surprised to learn that Twin B's loss was almost the same as Twin A's (Fig. 3). The hearing of both girls was tested again in February of 1954, and it was essentially the same as it was in November of 1953.

In the Denver public school system, auditory training and rehabilitation courses are available from the preschool age of 3½ years through high school. This program was started in 1926 and is conducted in a

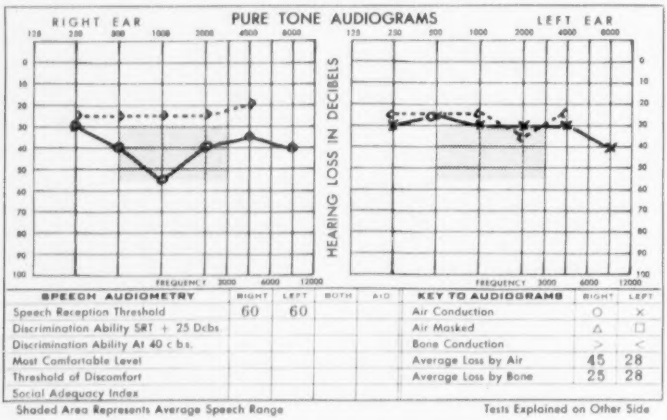
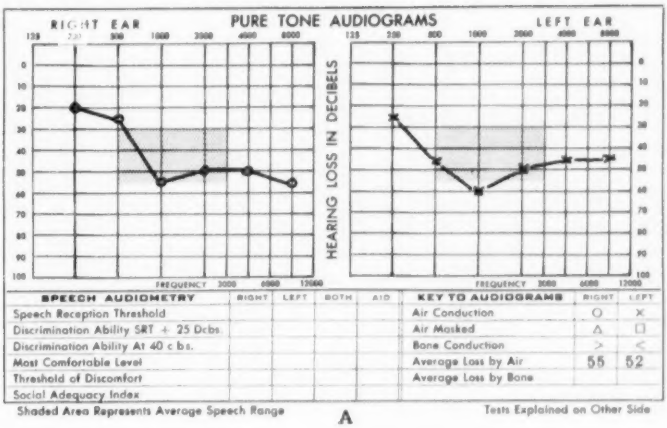


Fig. 2.—Audiograms of Twin A taken on March 14, 1953, showing improvement in the left ear by pure-tone tests, which, however, are incompatible with the speech reception threshold.



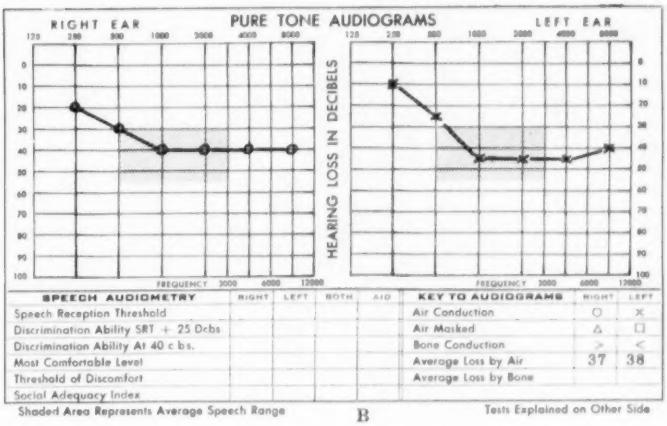


Fig. 3.—Results of hearing test made on Nov. 6, 1953. *A*, audiograms of Twin A; *B*, audiograms of Twin B.

regular school so that the children with hearing losses have some of their training with children whose hearing is normal.

Both girls were started on the auditory rehabilitation program in the fall of 1954 at the age of 7. They were both given used hearing aids which had been donated to the school and reconditioned by the hearing aid dealer. Each girl had a different make of aid. At first they were allowed to use the aids only at school under the supervision of teachers trained in auditory rehabilitation. After they had learned to use them they were allowed to use them at home and at play. Both wore them most of the time.

During Christmas vacation of 1954 we had the opportunity to retest their hearing. At this time they had each received three months of training in the use of their hearing aids. However, Twin B was getting a good result with her aid, but Twin A did not hear as well with her aid as she did without it (Fig. 4). When this was discovered Twin A was retested using Twin B's aid. Her speech reception threshold was improved from 70 to 35 db. Her discrimination score was reduced from 64% to 52%, probably because she was not used to the different frequency pattern of Twin B's aid.

When school started again after vacation Twin A was given another reconditioned aid, this time properly fitted. With this aid she had a speech reception threshold of 25 db., a discrimination score of 56%, and a social adequacy index of 46. Since the previous auditory training had been ineffectual due to an improperly fitted aid, even though her speech reception threshold with the new aid was the same as that of Twin B, her discrimi-

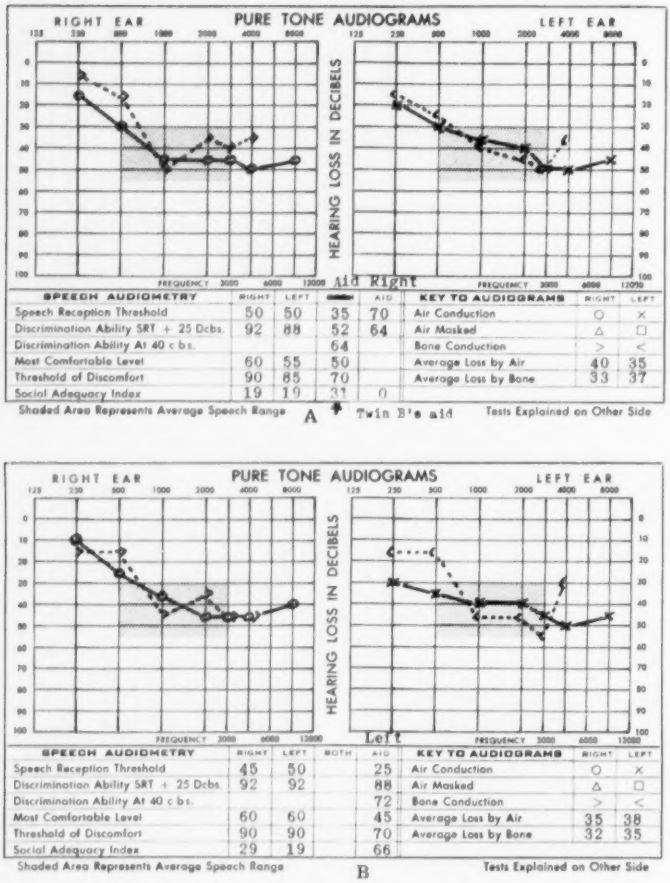


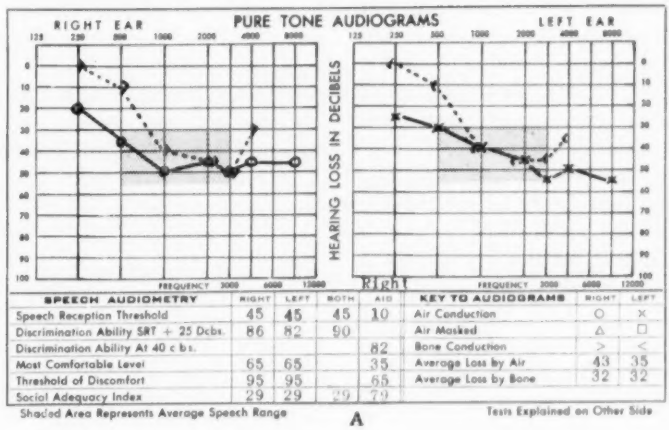
Fig. 4.—Results of hearing test made on Dec. 27, 1954, indicating that Twin B was getting a good result with her aid although Twin A was not. *A*, audiograms of Twin A; *B*, audiograms of Twin B.

nation score was only 56% as compared with Twin B's 88%.

Both girls continued their auditory rehabilitation programs for the remainder of the school year and were partially retested in the summer of 1955. At that time they both had identical results with their hearing aids. The speech reception threshold was 25 db., the discrimination score was 70%, and the social adequacy index was 57. These tests were purposely done with the volume of their aids set where they usually wore the aid rather than at a most comfortable level to listen to the material at 40 db. At this volume the discrimination score was not so good as before, so they were advised to wear the aids with more volume. Since they seemed to be so well adjusted to their aids they were allowed to return to their regular school.

In May of 1956 we retested both girls to decide whether they could continue in their regular school or should return for additional auditory training. The results of these tests were very similar, and both girls were doing better with their aids than previously (Fig. 5).

When this material was brought to the attention of the administrators of the Denver public schools they immediately agreed that their previous limited program of puretone threshold testing without speech testing was inadequate. A new complete two-room testing unit was installed, and they are now routinely doing complete speech and puretone audiometry on all children with hearing handicaps. In the first school year that this new two-room testing installation was used,



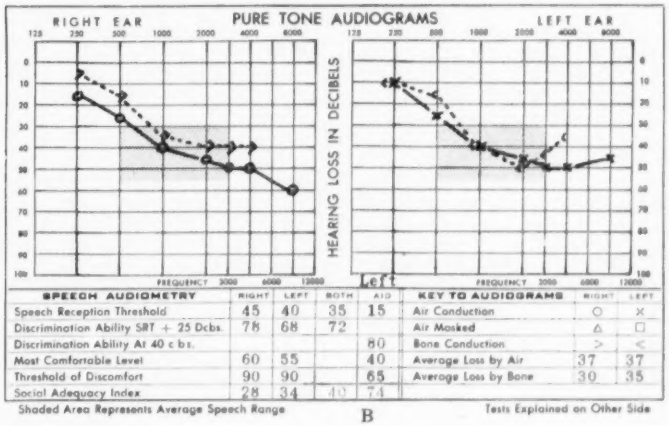


Fig. 5.—Results of hearing test made on May 14, 1956, showing improvement in both twins with the use of their aids. *A*, audiograms of Twin A; *B*, audiograms of Twin B.

79 children who wore hearing aids were tested by one of us (L. A. C.). Of these, 65% were advised to make some change in their aid. The advice given ranged from changes in the method of wearing the aid to the purchase of a new hearing aid. A program has been started to evaluate the results obtained by the children with hearing aids, at the start and finish of each school year. This study may give additional information about the importance of proper hearing aid fitting and auditory training in how to use the aid.

We have described an economical way to adapt an ordinary office in order to do speech testing.[2] It is not a complicated test, and a secretary or a nurse can do it with more accuracy than they can do pure-tone audiometry. Since patients continue to ask their physicians about hearing aids, otolaryngologists should equip themselves to give good and adequate advice about the many problems involved in the use and selection of a hearing aid.

Summary and Conclusions

Identical twin girls who had enough hearing to learn to speak normally and clearly developed almost identical bilateral deafness. They had received hearing aids and special auditory training for three months when it

was discovered that Twin B heard well with her aid but Twin A did not hear as well with her aid as she did without it. When Twin A used Twin B's aid she did much better. Twin A was properly fitted with another aid, and after six months of additional auditory training she was making as good use of her hearing aid as was her twin sister.

Since the hearing losses were so similar and the final results with their aids are almost identical, we feel that the poor results as shown by Twin A were due to an improperly fitted hearing aid. The improvement in Twin A's hearing with a properly fitted aid, after she learned to use it, as compared with the results when she first received it, proves the necessity of auditory training in learning to use a hearing aid.

Since an economical method of speech testing, using one's present office space, is available, the otolaryngologist should equip himself to advise his patients about the uses and abuses of hearing aids.

612 Republic Building (2).

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Relative Effectiveness of Three Modes of Delayed Sidetone Presentation

RICHARD A. WINCHESTER, M.A., and EDWARD W. GIBBONS, M.A., Los Angeles

Previous research has demonstrated the theoretical and clinical significance of the speech distortions associated with binaural presentation of delayed sidetone. Changes in vocal intensity, quality, and pitch, as well as deterioration in articulatory patterns resulting from delayed sidetone interference, have been investigated and reported in detail.

It has been established that an amplified sidetone having a signal delay ranging from 0.14 to 0.18 second can produce maximal disruption of speech production,[1] and these changes can manifest themselves with delayed sidetone presentation levels as low as 20 db. re subject threshold for speech.[2] At higher presentation levels, e. g., 40 db. and upward, voice and articulatory dysfunctions become increasingly spectacular and disruptive to oral communication.

Although quantification of these alterations in vocal and articulatory functions has been investigated, as yet the most readily measured effect of delayed sidetone interference with speech is a single resultant of the combined voice and articulatory distortions—the retardation of the normal reading rate.

These objective changes in speech patterns have formed the basis for the application of delayed sidetone techniques to the problem of the detection and quantification of nonorganic hearing deficits.

Hearing losses of varying degrees of binaural asymmetry are frequently encountered in medicolegal audiometric evaluations. Whenever the subject is unwilling or unable to demonstrate accurate subjective thresholds binaurally, traditional tests, such as the Stenger test, can fail to detect a functional, uniaural component.[3] Newer methods such as delayed sidetone techniques depend upon the voluntary responses of the subject to a minimal degree. As a result, delayed sidetone techniques promise to become a valuable addition to the clinical audiologist's test armamentarium in the area of forensic audiology.

Before complete reliance can be placed upon delayed sidetone testing, questions regarding adaptation,[4] fatigue, and mode of presentation must be answered. This need for information has been indicated by the problems which have arisen during the exploratory application of delayed sidetone techniques in several hundred cases showing significant degrees of nonorganic hypacusia. These cases were routinely examined by clinical audiologists, whose test results form the basis upon which medicolegal adjudicative ratings for hearing disabilities are assigned.

This investigation is concerned with the demonstration of the relative effectiveness of several modes of delayed sidetone presentation: binaural, uniaural, and uniaural with contralateral masking. It was presumed that this information should solve certain problems regarding procedures for testing ears separately using delayed sidetone.

Subjects

One hundred sixty normally hearing adults with speech reception thresholds for each ear of 5 db. or better were selected for testing from among veterans seen at the Veterans Administration Regional Office at Los Angeles. All persons considered for the investigation were checked for reading ability and screened to exclude neuropsychi-

Accepted for publication Sept. 11, 1956.

atric disabilities and speech or language deviations.

Equipment

The sidetone delaying equipment was the Magnecorder Tape Recorder, Model PT-63AH, with the PT-63J amplifier. This unit has been permanently wired into the U. S. Recording Hearing Evaluator, Model 200, making it possible to introduce monitored delayed sidetone uniaurally or binaurally at designated sensation levels through Channel I. Through Channel II of the hearing evaluator it was possible to present saw-tooth wave-form masking noise at designated sensation levels to the subject. Permaflux receivers, Model PDR-8, with MX41AR cushions were used in all tests. Input to both channels was aligned daily against known intensity levels, and output to the headphones was calibrated weekly. The testing equipment was located in a standard two-room test suite.

The prose passage used to elicit oral reading responses was taken from a fifth-grade reader currently employed in the Los Angeles public schools. The 1950 census figures indicate that 91% of the adult population in the U. S. has completed the fifth grade. By choosing materials at this level of reading difficulty, it was anticipated that few of the subjects screened for reading ability would be rejected. This passage contained 500 syllables of expository prose which had been demonstrated to be homogeneous with respect to reading difficulty.

Test Procedures

Four different test conditions were arranged, and each person studied was subjected to a single test condition. Thus, problems arising from adaptation to reading materials or to the delayed sidetone were minimized. The four test conditions were those presented in Table 1.

In Condition A, the test passage was read under delayed sidetone binaurally presented at 60 db. sensation level (SL). Condition B involved the reading of the same passage under a 60 db. SL of delayed sidetone directed through the same phone to every subject. The opposite ear was unmasked. In Condition C, each subject read the common prose passage under 60 db. SL of delayed sidetone directed in every case through the same phone to the subject, while the opposite ear was subjected to approximately 80 db. SL of saw-tooth wave-form masking noise. Condition D involved the reading of the test passage without experimentally induced delayed sidetone or masking noise. This last condition was set up for comparison purposes; from it the unimpeded reading rate was inferred. Sidetone delay was 0.16 second in every case. Standard reading instructions were given to each subject to read naturally and in no event to stop, or in any way to change deliberately the normal delivery of speech.

The sensation level of 60 db.[5] for the delayed sidetone was chosen because it would have a marked inhibitory influence upon the speech of the test subjects while not making it impossible to mask during the uniaural masked test condition. The sensation level of 80 db.[6] for the masking noise was chosen because it did not appear to interfere through auditory cross-over with the functions of the ear under delayed sidetone stimulation during the masked uniaural test condition.

TABLE 1.—*Modes of Delayed Sidetone Presentation*

Test Condition	Mode of Presentation	Sensation Levels		Sidetone Delay Time
		Sidetone	Masking	
A	Binaural sidetone	60 db.	0 db.	0.16 sec.
B	Uniaural sidetone only	60 db.	0 db.	0.16 sec.
C	Uniaural sidetone + masking	60 db.	80 db.	0.16 sec.
D	No induced sidetone	0 db.	0 db.	

TABLE 2.—*Group Reading Times in Seconds*

Test Condition	N	Mean Age	Males, %	Mean Time	Standard Deviation	Range
A	40	37.6	75.0	179.8±3.8	24.4±2.8	130.6-238.2
B	40	32.9	85.0	160.3±3.0	19.1±2.2	129.0-209.4
C	40	39.2	75.0	169.8±4.2	26.4±3.0	132.7-223.6
D	40	31.6	77.5	129.8±3.4	21.3±2.4	90.5-183.8

Results

Variations in response to the three different models of delayed sidetone presentation were measured in terms of temporal variations. Attempts to objectify and to measure other variations in the speech of the subject were abandoned because of the difficulty in recording and analyzing these factors. Table 2 summarizes the data related to the distributions resulting from the four test conditions.

Reference to Table 2 will show that the mean reading times for the four groups varied and that, in terms of ranges of scores and standard deviations, each group showed a dispersion from group means. Thus it was inferred that there were possible differences in performance within all groups and from group to group. All groups appeared to be similar with respect to mean ages and the percentage of males.

When analyses of variance were obtained from the four distributions as a means of demonstrating the significance of the differences in group reading times (Table 3), it was shown that the reading times under the binaural mode of delayed sidetone presentation (Condition A) differed very significantly from the unimpeded reading time (Condition D). In addition, it was found that the reading time for the uniaural mode of presentation without contralateral masking (Condition B) differed very significantly from the unimpeded reading rate (Condition D). The reading time for the uniaural mode of presentation with contralateral masking (Condition C) also differed very significantly from the unimpeded reading time (Condition D).

When analyses of variance were computed for the three different modes of delayed sidetone presentation, it was seen that the binaural mode of presentation (Condition A) and the uniaural mode of presentation without contralateral masking (Condition B) differed very significantly. Moreover, the reading times for the two uniaural modes of presentation, without contralateral masking (Condition B), and with contralateral masking (Condition C), differed significantly. Finally, the reading times for the binaural mode of presentation (Condi-

TABLE 3.—*Analysis of Variance for Testing Differences in Intergroup Oral Reading Times*

Group Comparison	Sums of Squares	Degrees of Freedom	Mean Squares	F Ratios	P
A to D	115.21 280.54	1 78	115.21 3.597	32.02	>0.01
B to D	378.46 208.74	1 78	378.46 2.676	141.43	>0.01
C to D	231.21 325.54	1 78	231.21 4.174	55.39	>0.01
A to B	76.06 407.74	1 78	76.06 5.227	14.55	>0.01
B to C	18.06 354.82	1 78	18.06 4.549	3.97	0.05
A to C	20.01 524.54	1 78	20.01 6.725	2.98	Nonsig.

tion A) and the uniaural mode of presentation with contralateral masking (Condition C) did not differ significantly.

Comment

Although the binaural mode of presentation proved to be very effective in producing variations in oral reading time, its use does not enable the audiologist to test ears separately. Consequently, whenever absolute or relative threshold values for both ears are indeterminate, the uniaural mode of presentation with contralateral masking is indicated.

Although the uniaural mode of presentation without contralateral masking produced significant changes in the oral reading time, this mode of presentation must not be used, since it can produce spurious results. The presence of even a relatively small amount of hearing sensitivity in the ear not under test, because of its speech monitoring functions, tends to produce a reduction in delayed sidetone inhibitory effects, thus erroneously suggesting a greater deficit of hearing in the ear under test.

The results of Condition C indicate the feasibility of testing ears separately. However, care must be taken to mask the contralateral ear adequately. Results bear out the original assumption that a masking noise sensation level of 80 db. is effective in cases of normal auditory acuity. However, sensation levels beyond 80 db. should be avoided whenever delayed sidetone sensation levels are no more than 60 db., because of the danger of shifting the threshold through auditory cross-over of the ear under sidetone stimulation.

Results of Condition D indicate that marked differences in oral reading times do exist even when materials used to elicit the oral reading responses are carefully chosen to avoid undue difficulty in reading. As a result of this, it is not possible to establish a standard unimpeded time against which sidetone test results may be evaluated. In other words, at this stage of development of these tests, the subject's unimpeded and impeded reading times must always be obtained. Care must be taken to minimize effects of adaptation and fatigue in the test situation by not using prolonged reading passages.

It is important to note that the masking process in delayed sidetone audiometry involves perceptual functions not elicited in standard threshold determination procedures in which the aim of the tester is to shift sufficiently the threshold of the ear not under test so that the external, contralaterally applied stimulus is not perceived. In the case of delayed sidetone audiometry the stimulus which must be masked is internally generated. The masking of the ear not under test must be accomplished, even though the thresholds of the two ears are the same.

Summary and Conclusions

One hundred sixty normally hearing adults tested under three different modes of delayed sidetone presentation and one condition of unimpeded reading in an effort to investigate the relative effectiveness of these different modes of presentation gave the following results:

1. The binaural mode of presentation is very effective, and should be used only when it is not necessary to test ears separately.

2. The uniaural mode of presentation without contralateral masking is the least effective, and should not be used, as it will yield inaccurate test results.

3. The uniaural mode of presentation with contralateral masking is as effective as the binaural mode of presentation, and can be used to test ears separately.

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Audiometric Examination of Parents of Children Deaf from Birth

The Influence of Consanguineous Marriage

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Among 139 marriages from which resulted 507 children, 187 of the children were born deaf ("rubella children," etc., excluded). These deaf children were all pupils of the Royal Institute for Deaf Mutes. In 17 of the marriages the parents were related to each other. They had 85 children, of whom 28 were deaf. In 11 cases the parents were full cousins (Families 1, 2, 3, 4, and 5), in 1 marriage twice full cousins (Family 6), in 1 full cousins and also second cousins, and in 4 second cousins (Family 7). These 17 consanguineous marriages represented 12.2% of the total 139 marriages; if we take into consideration only the 13 first-cousin marriages, the proportion is 9.4%, with a standard deviation of $\pm 2.5\%$. In the entire population of Holland, the proportion of consanguineous marriages is about 0.16% (Polman [1]).

Earlier investigators found proportions of 6.47% (De Wilde [2]) of consanguineous marriages from the census of 1909, including all forms of deafness, and 12.2% (Woltjer [3]) from among congenitally deaf pupils of the Groningen Institute in 1931. My percentages agree with values found in other countries. For example, in Denmark Lindenov [4] in 1945 noted 9.3% of congenitally deaf children with consanguineous parents, and about 2% of consanguineous marriages in the entire population. In Northern Ireland (Stevenson and Cheeseman,[5] 1956), similar figures were 6.1% and 0.1%–0.4% (first-cousin marriages). Hopkins and Macklin [6] (U. S. A.) in 1946 gave the figures 8% and 0.2%. In addition to these data, I shall mention Johnsen [7] (Denmark), who in 1952 found 6 consanguineous marriages (8.3%) among 72 marriages resulting in children with partial high-frequency deafness, most of them having a recessive progressive type.

Most geneticists accept the cause of the recessive hereditary form of congenital deafness to be a monomeric (one-gene) transmission, though pedigrees have been described which make it likely that several recessive genes, independent of each other, may be responsible for deafness. In cases of recessive heredity we may expect that the parents (phenotypically normal, i. e., with good hearing) of the affected child (i. e., deaf) are heterozygotes for the gene "deafness."

"Heterozygous" means that a person has a single gene for a certain quality; a "homozygous" person possesses it twice (from the father as well as from the mother). A recessive quality manifests itself only when it is homozygous. Two heterozygous parents have children with a homozygous recessive trait at a ratio of 1:4. Related parents are more frequently both heterozygous for some gene, i. e., for deafness, than are nonrelated parents, as they both have a great chance to inherit the gene from one of their common ancestors. That is the reason we may expect among children with some recessive illness, i. e., deaf-mutism, relatively more instances of related parents (cousins) than occur in the population as a whole. If there is a significant difference in the proportion

Accepted for publication June 4, 1956.

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of consanguineous marriages among parents with children having an illness and the proportion of such marriages among the entire population, there is a great chance that the illness in question is a hereditary one. The figures on deafness mentioned above point strongly to heredity.

It is known in several recessive illnesses that by means of subtle, specialized examination apparently completely normal persons are detectable as heterozygotes; that is, they show slight aberrations related to the illness of the manifestly ill, homozygous person. This is the case in albinism and in hemophilia. Now it would be of great importance, both scientifically and practically (in genetic counseling), if apparently normally hearing persons who might be heterozygous for deafness could be detected as carriers of the recessive gene.

Some investigators have tried to solve this problem. Tinkle [8] (U. S. A.) in 1933 examined with a 2-A audiometer three parents of deaf children. They showed no important impairment of hearing. Concerning the audiograms of the deaf children, he gave no details. Secrétan [9] (Switzerland) in 1954 examined audiometrically nine parents from families comprising some congenitally deaf relatives. Eight of them had an audiogram too low in the high frequencies, and some of them showed traumatic or normal presbycusis. Four of them, however, were 60 years of age or older. Two of these parents had been tested, but not by audiometer, about 10 years earlier, and at that time their hearing was still "normal." The ninth parent showed a normal audiogram. Another group of parents was examined only with tuning forks.

Lindenov [4] (Denmark) in 1945 made some examinations in the "reverse" sense: He examined the 11 children of 6 parents who presumably were hereditarily deaf and whose husband or wife had normal hearing. The children, who must have been heterozygous, had normal hearing. Lindenov did not mention the values on their hearing tests, but they were "normal." Tinkle [8] in 1933 examined a child, presumably heterozygous, having a loss of hearing of only 15%-0%. Stevenson and Cheeseman [5] (Northern Ireland) in 1956 stated in their paper (p. 194): "Where, by reason of the condition of the parents, hearing offspring must have been heterozygous for one gene or for two separate genes, in all subjects tested the audiograms were normal." They gave no details of their audiograms. For the sake of completeness, I shall mention Johnsen [7] (Denmark), who in 1952 made a family investigation on severe hardness of hearing, examining some parents of children presumably having recessive progressive hardness of hearing. He gave no details of the audiograms of the parents, but they were said to be "normal." Furthermore, Johnsen [10] in 1954 examined six consanguineous sets of parents, each having a child with a progressive hearing impairment. They were "normal" ("as demonstrated by objective examination"). It is not quite clear to me to which form of hearing impairment his "parent examinations" referred.

All these authors came to the conclusion that there are no typical aberrations in the audiograms of parents or (heterozygous) children that may distinguish them as heterozygous.

Present Study

As the audiometric examinations mentioned in most investigations are not very extensive, and while data on the audiograms are sparse, I studied the problem in a group of parents, giving full details concerning the audiograms and pedigrees. Vestibular reactions were not tested, as irritability of the vestibular organ not only cannot be considered a good differentiation between hereditary and acquired deafness (Ulrich and Corrodi-Sulzer [11]) but in presumable heterozygotes certainly will not answer the question whether an eventual impairment of hearing may be hereditary.

First, a number of pairs of consanguineous parents were tested audiometrically, as it may be expected that cousins having con-

genitally deaf children are heterozygous, as stated above. So much the more is this the case if they have other deaf relatives. In addition to these parents, a number of parents were examined who were not related to each other, who had other deaf children, and in whose families other deaf persons occurred, so that heredity was very probable. Of course, the principle is the same: The parents have a great chance to be heterozygous for deafness.

The audiometric examinations were performed with a Pedersen audiometer, which can be regulated continuously from 125 to 16000 cps in 1 db. steps of intensity. The results were criticized by Prof. H. C. Huizing, of the Audiological Department of the Ear-Nose-Throat Clinic of the University (Director, Prof. Eelco Huizinga), in which department the audiograms were made.

The following audiograms with pedigrees are the result of my investigation. I shall give only a short description of them, as the figures speak for themselves. The audiograms of the parents are bordered in order to mark them distinctly.

Results

I. Consanguineous Marriages

Family 1 (five deaf children).—Parents had normal audiograms, as did one of the normally hearing children (III).

Family 2 (one deaf child).—Parents had normal audiograms.

Family 3 (one deaf child).—Father showed a dip at 4000 cps for both ears, may be the result of trauma (noise). Mother had a normal audiogram.

Family 4 (one deaf child).—A sister had amaurotic familial idiocy; two cousins, also the children of a full-cousin marriage, were mentally retarded, one of them having congenital vitium cordis and polydactyly. Father of the deaf child may have presbycusis. Bone conduction as air conduction. Mother's audiogram was normal.

Family 5 (one deaf child).—Father had normal hearing. Mother's audiogram was somewhat too low in the high-tone range.

Family 6.—Parents of child II were double first cousins. Father showed a dip at 4000 cps for both ears, probably of traumatic origin. Mother's audiogram was normal. Parents of child V: Father had a dip at 4000 cps (traumatic?). Mother's hearing was normal.

Family 7.—Parents were second cousins. Father showed severe loss of hearing in the high-tone range, probably congenital. Bone conduction as air conduction. Mother had a slight loss of hearing for high tones in the right ear. Bone conduction as air conduction.

II. Parents Not Cousins, but in Each Marriage More Than One Child Congenitally Deaf.

Family 8.—First marriage: Father's hearing was normal. Mother showed questionable precipitated presbycusis. Bone conduction as air conduction.

Second marriage: Father had a dip at 4000 cps for the left ear, must be acquired. Mother showed a dip at 8000 cps for the left ear, must be acquired also.

Family 9.—Parents' audiograms were normal.

Family 10.—Father and mother showed a dip at 4000 cps, must be acquired.

Family 11.—Father had loss of hearing caused by otitis media duplex. Bone conduction normal. Mother's hearing was normal.

Family 12.—Father showed questionable presbycusis in left ear. Curiously, he was completely deaf in the right ear. He was not aware of it! Cause unknown. Mother showed a dip at 4000 cps of possible traumatic origin.

Family 13.—Two boys had a severe hearing impairment. Parents' audiograms were normal.

Though many parents showed some impairment of hearing, e. g., dips, only one parent (Family 7, I) had what was most probably a congenital loss of hearing.

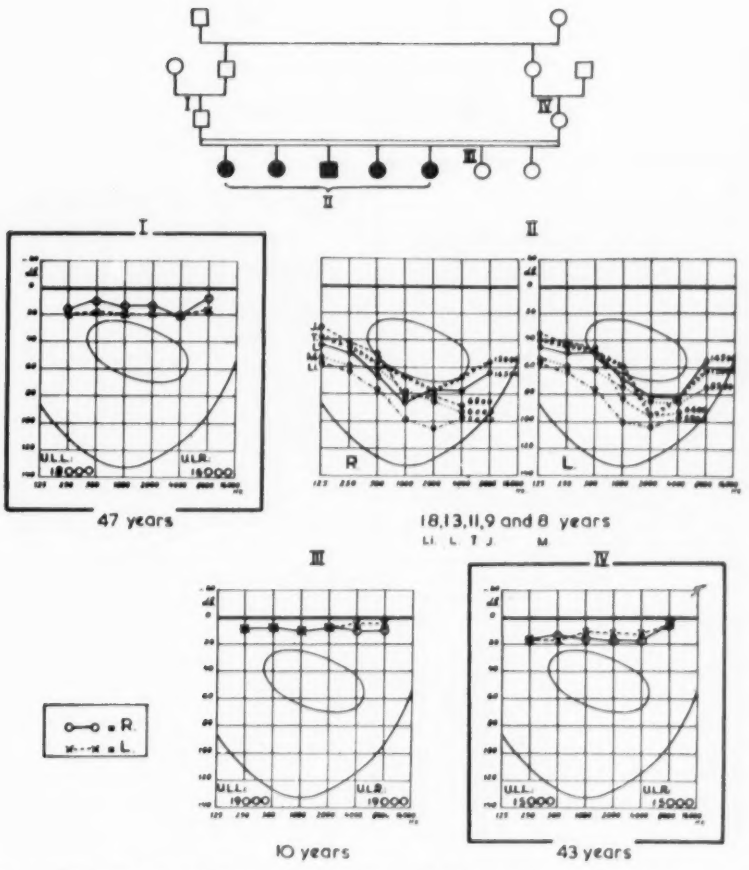

Fig. 1.—Pedigree and audiograms of Family 1. In this and the following figures, shaded symbols indicate congenitally deaf children. Bordered audiograms are those of the parents.

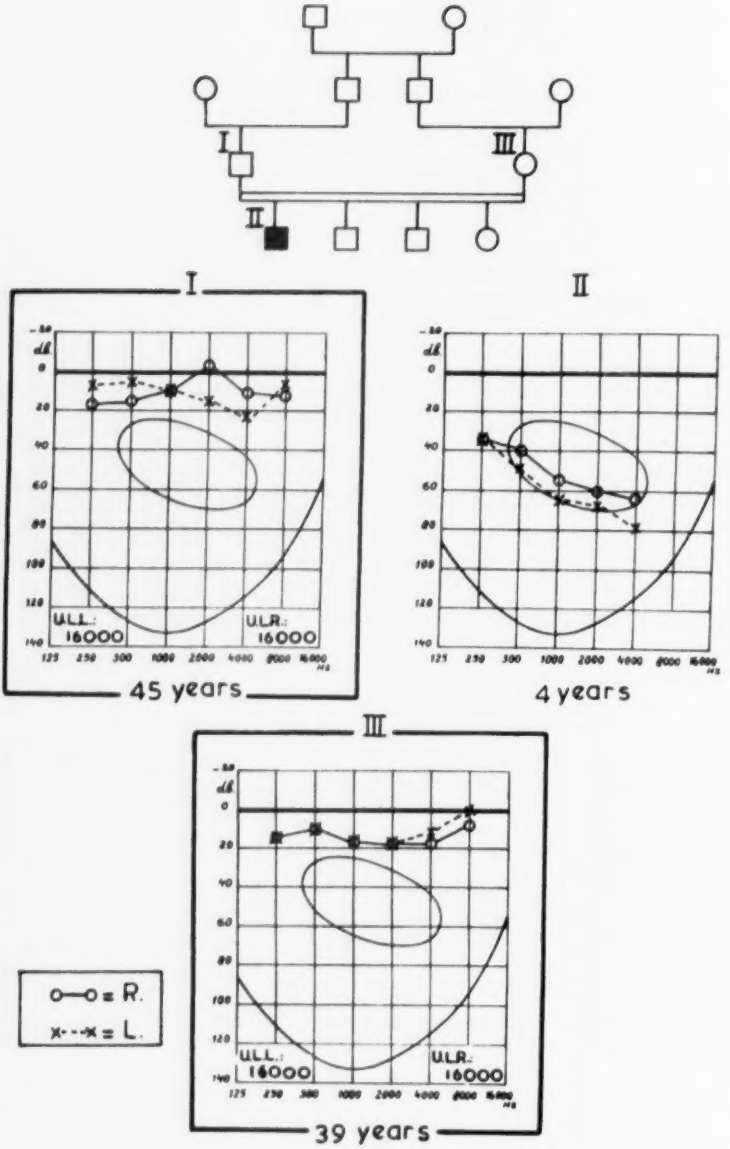

Fig. 2.—Pedigree and audiograms of Family 2.

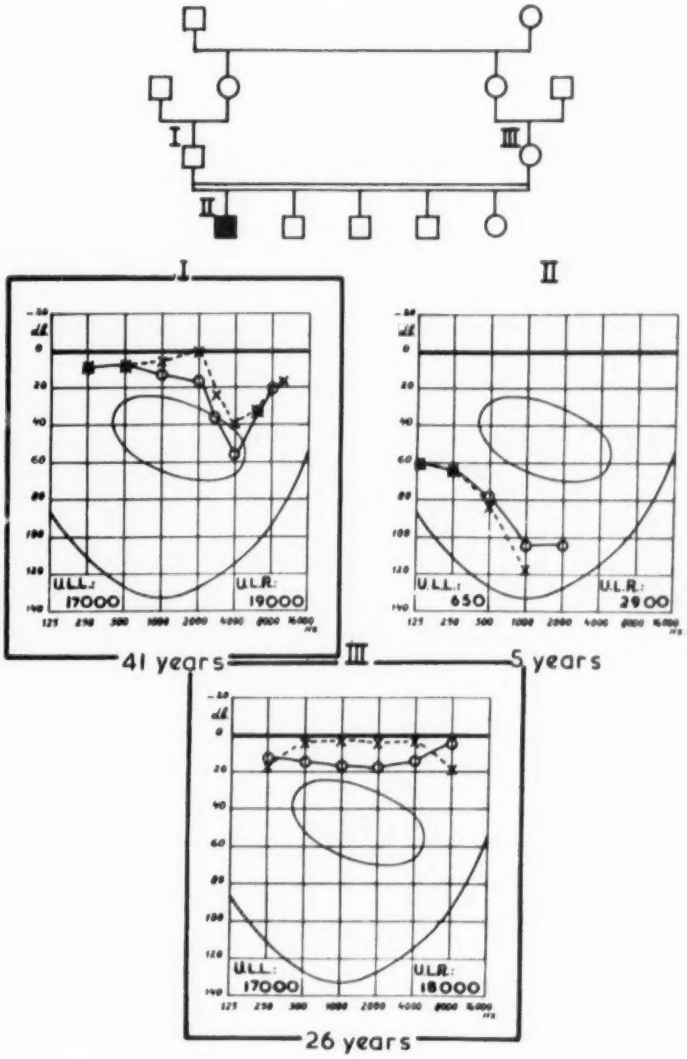

Fig. 3.—Pedigree and audiograms of Family 3.

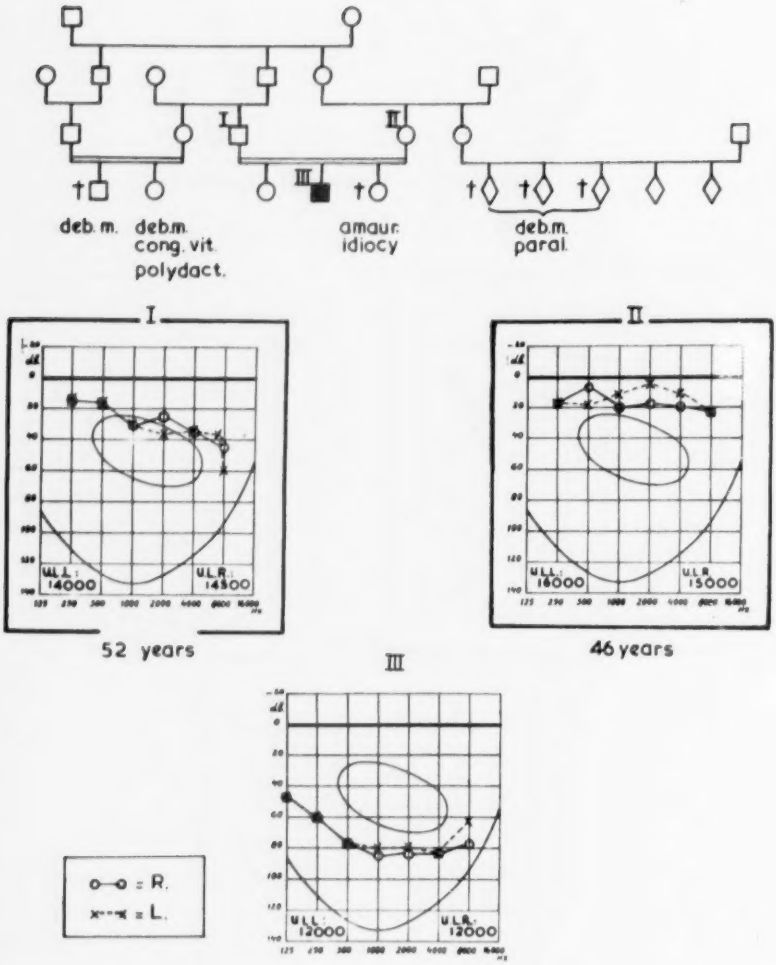

Fig. 4.—Pedigree and audiograms of Family 4.

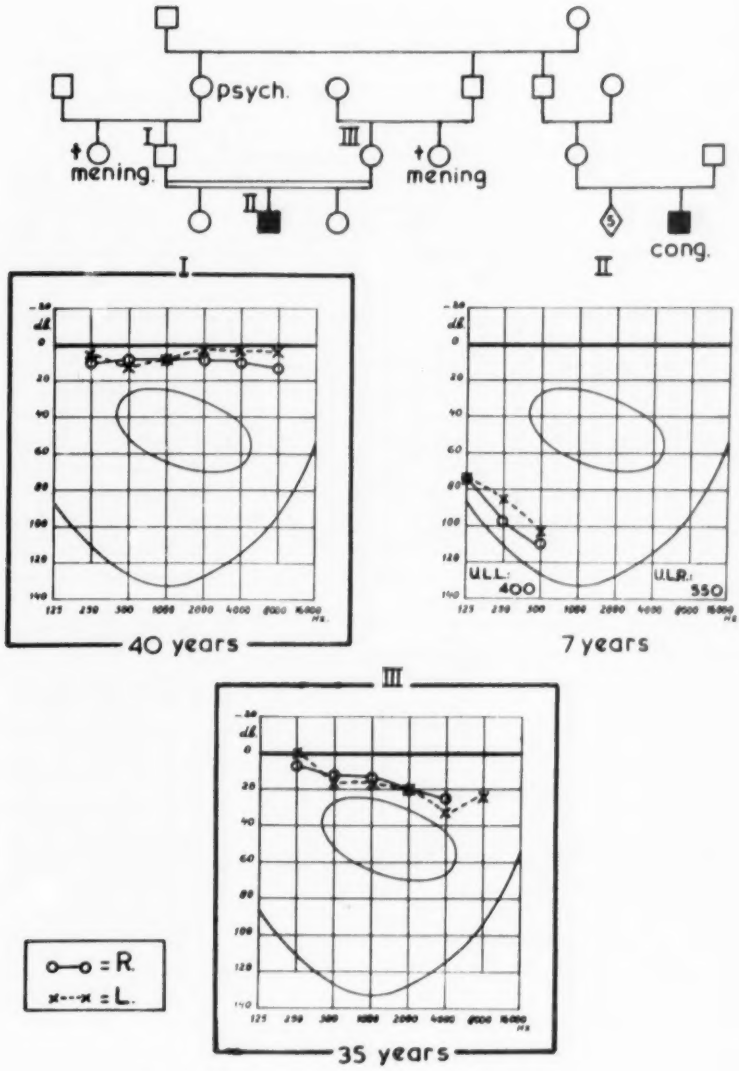

Fig. 5.—Pedigree and audiograms of Family 5.

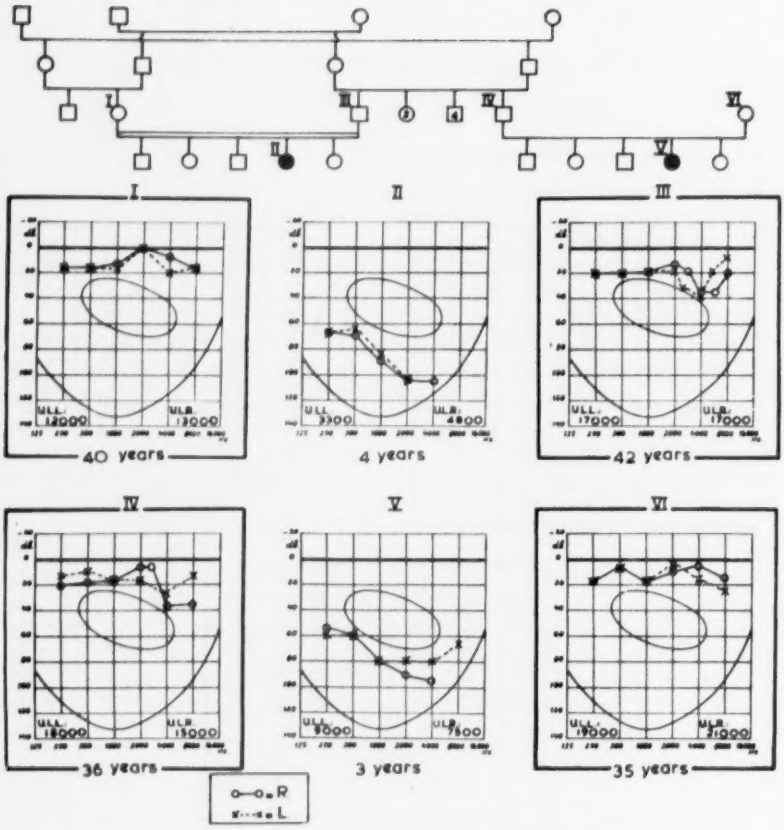

Fig. 6.—Pedigree and audiograms of Family 6.

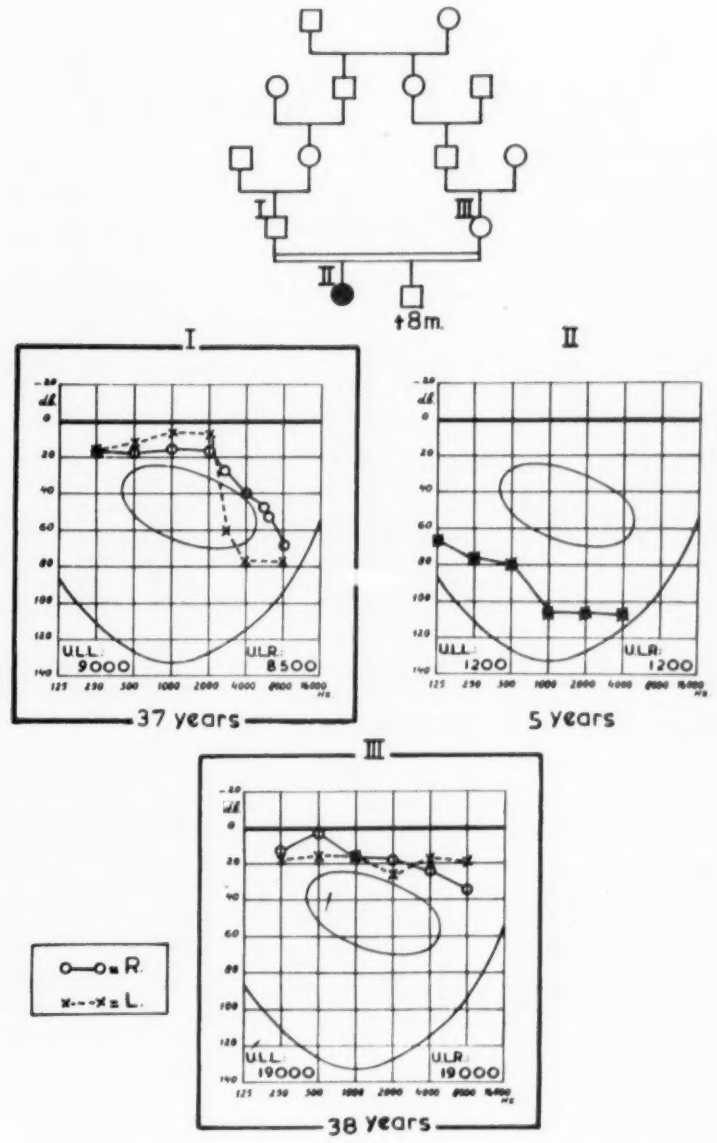

Fig. 7.—Pedigree and audiograms of Family 7.

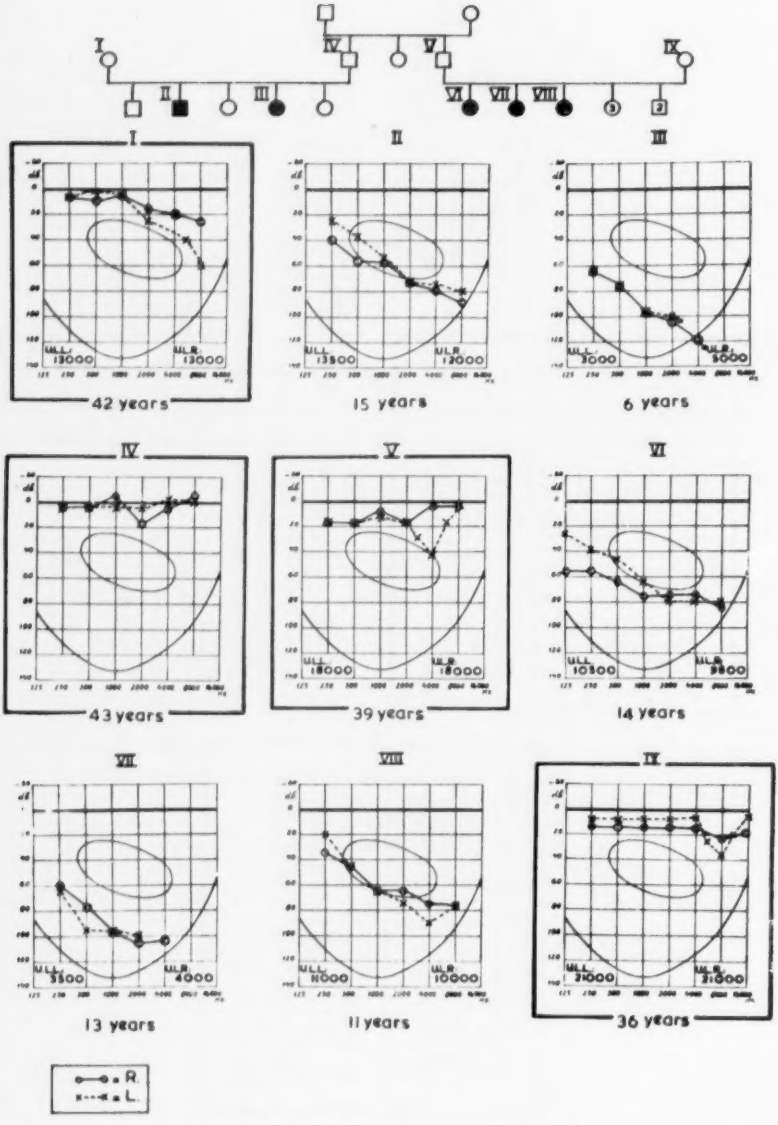

Fig. 8.—Pedigree and audiograms of Family 8.

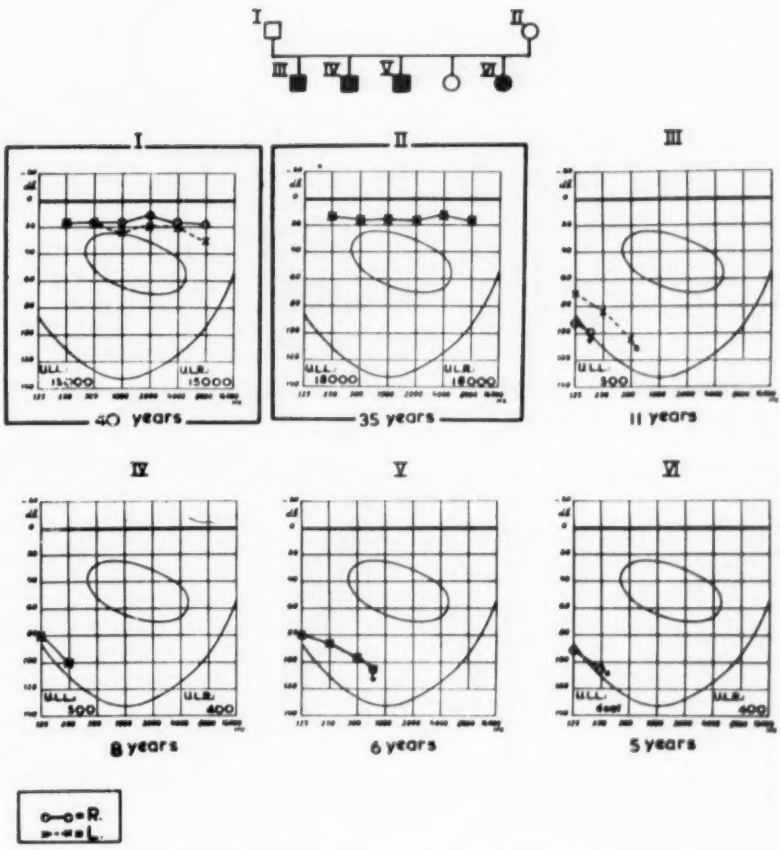

Fig. 9.—Pedigree and audiograms of Family 9.

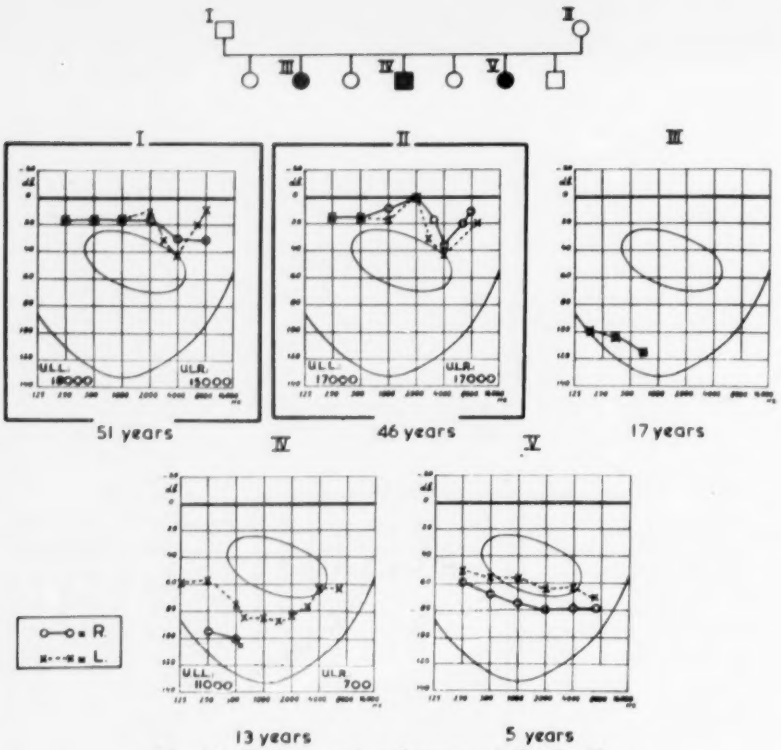

Fig. 10.—Pedigree and audiograms of Family 10.

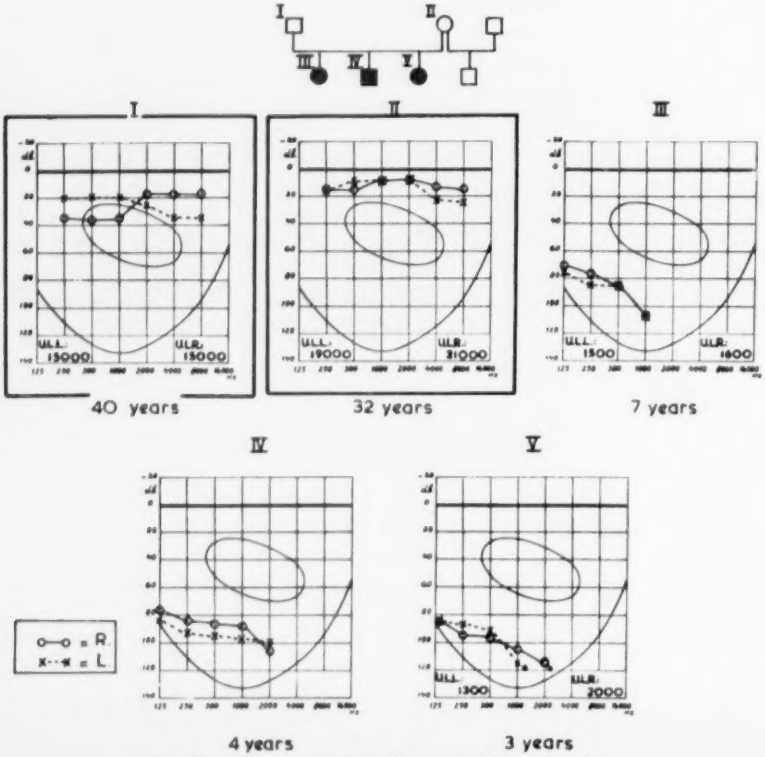

Fig. 11.—Pedigree and audiograms of Family 11.

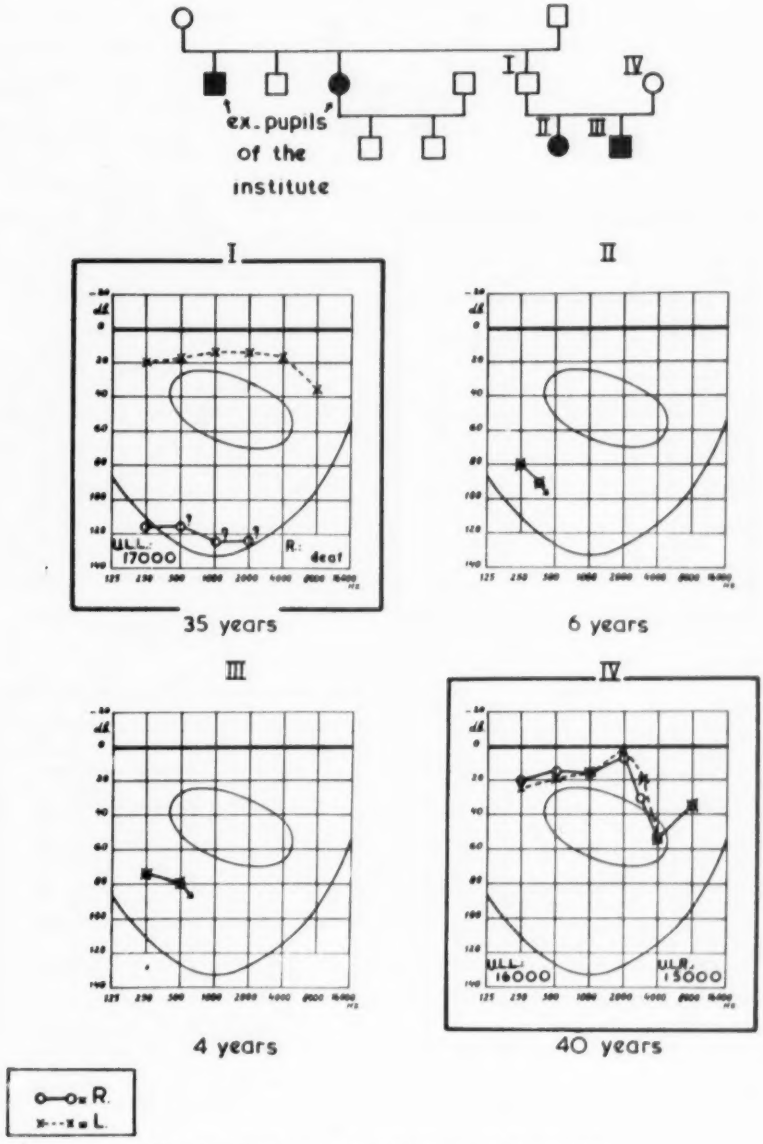

Fig. 12.—Pedigree and audiograms of Family 12.

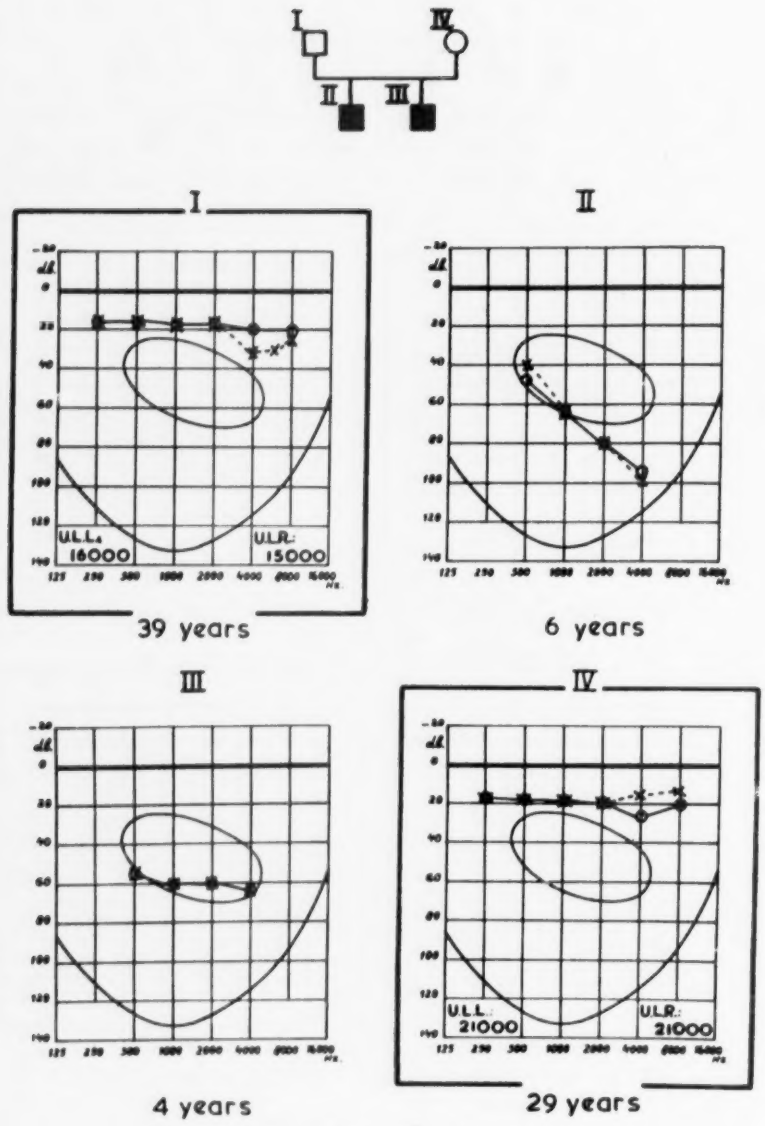

Fig. 13.—Pedigree and audiograms of Family 13.

Comment

The great difficulty in a study such as the present one is that so many causes may change the hearing acuity. Slight, or even severe, traumata may be unknown and alter the threshold audiograms. If, for example, a dip is suspected to be of traumatic origin, we hardly ever know at what age or time the trauma occurred and the dip appeared in the audiogram. Most otologists consider a dip a result of exposure to the influence of noise (e. g., Johansen [12]). Theoretically, however, we cannot even say with certainty that the dip was not congenital! Furthermore, there may be a precipitated presbycusis (see in connection with this, e. g., the extensive investigation on "Hearing Reduction Caused by Advancing Age" by Johansen [12]). We also have to take into account that even in young children there may be an abnormal loss of hearing for the high frequencies which may increase with age (Guild [13]). It is not impossible that precipitated presbycusis appears more frequently in heterozygotes. It is even conceivable that "heterozygous ears" are more susceptible to acoustic traumata than "homozygous normal ears," but these are only suppositions.

The conclusion of my entire investigation is that normally hearing parents of children with recessive deafness are not detectable as heterozygotes. Moreover, as a result of my investigation and of those of the authors mentioned, and taking into account the difficulties discussed above, I think it is impossible to solve this problem audiometrically.

Summary

I examined 15 sets of presumably normally hearing parents and their 32 congenitally deaf children, who probably had recessive hereditary deafness, in order to see whether these parents were detectable as heterozygotes. In seven cases the parents were related to each other (cousins). Threshold audiograms of them and of their deaf-mute children are presented. The parents turned out not to be detectable as heterozygotes. The importance of consanguinity is emphasized.

Prof. H. C. Huizing, of the Audiological Department of the Ear-Nose-Throat Clinic, University of Groningen, read the manuscript critically.

Hoge der A 10.

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Clinical Notes, New Instruments and Techniques

A Method of Tracheotomy Care

FRANK C. MARCHETTA, M.D.; SIDNEY ANTHONE, M.D., and ANNA AUNGST, R.N., Buffalo

In recent years, the indication for tracheotomy has been recognized more frequently. Severe accident cases, chest injuries, postoperative neurosurgical cases, and head and neck cases often require tracheotomy, not only to prevent immediate asphyxiation but also to promote a smoother post-traumatic or postoperative convalescence.

The successful outcome of many of these cases is partially dependent on a systematic and efficient system of tracheotomy care. Experience has proved that this is often lacking on some surgical floors.

Many of the patients admitted to the Head and Neck Service of the Roswell Park Memorial Institute require tracheotomy. It is the purpose of this paper to describe a simple and adequate method of tracheotomy care now in use on this Service.

The method of care must take into consideration the alterations in physiology of the patient. First, the freshly tracheotomized patient is unable to cough efficiently and rid the tracheobronchial tree of secretions. This necessitates tracheal aspirations by an attendant. Second, these patients are unable to expectorate and secretions will accumulate in the oropharynx and hypopharynx. Third, oral hygiene must be provided, especially in patients who are comatosed or who have undergone intraoral surgery. Fourth, the tracheotomy bypasses the nasal cavity and accessory sinuses, which act to humidify the air, and undue drying and crusting within the tracheotomy tube and the trachea may occur. Fifth, cleanliness and sterility must be maintained.

The basic equipment includes items which are readily available in any hospital: (1) a metal tray approximately 12×15 in., (2) four empty 500 cc. Baxter or Abbott blood bottles, (3) two No. 14 whistle-tip catheters, (4) two Asepto syringes with a short length of soft rubber tubing attached to the tip, (5) a package of 4×4 sponges, (6) a roll of 1 in. adhesive tape, and (7) an efficient suction apparatus (Fig. 1).

One of us (F. C. M.) designed a special piece of apparatus for holding an aspirating catheter firmly in its bottle. This consists of a band of spring steel which will snap onto a 500 cc. clysis bottle. A small spring clip, into which the catheter can be slipped, is fastened to the center of the metal band (Fig. 1). Loops made from adhesive tape, however, serve the same purpose (Fig. 2).

The four bottles are arranged on the tray. The first bottle is equipped with a metal holder and contains aqueous benzalkonium (Zephiran), 1:10,000. A No. 14 whistle-tip catheter is inserted into the bottle, and the proximal end is fastened into the clip of the metal band holder. This bottle is labeled "Tracheal Suction."

The second bottle is also equipped with a metal holder and also contains 1:10,000 aqueous benzalkonium. A catheter is similarly placed as in the first bottle, and this bottle is labeled "Oral Suction."

The third bottle contains a solution of 50% peroxide and 50% water. An Asepto syringe, with a short length of soft rubber tubing attached to its tip, is inserted into this bottle.

Accepted for publication Aug. 22, 1956.

From the Head and Neck A Service of the Roswell Park Memorial Institute. (Acting) Chief Cancer Research Head and Neck Surgeon: Dr. Marchetta.

Fig. 1.—Basic equipment necessary for tracheotomy care. The metal clip, for holding a catheter in its bottle, is shown in the center of the photograph.

Fig. 2.—Tracheotomy set-up with the use of adhesive tape loops instead of metal clips for holding catheters in place.

The fourth bottle contains isotonic saline and is also equipped with an Asepto syringe.

A container of sterile 4×4 sponges is kept on the tray (Fig. 3).

The freshly tracheotomized patient, as a rule, needs frequent suctioning. When tracheal suctioning is necessary, the catheter is removed from the bottle, attached to a suction apparatus, and the secretions aspirated from the tube and trachea (Fig. 3). When suctioning is complete, water is sucked through the catheter to rid it of secretions, the outside of the catheter is wiped clean with a sterile 4×4 sponge (Fig. 4), a small amount of benzalkonium solution from the tracheal suction bottle is aspirated through the catheter, and the catheter is detached from the suction apparatus and reinserted into the proper bottle. We insist that the catheter be detached from the suction apparatus because the weight of the suction tubing may drag the catheter from its bottle onto the floor.

When oral suctioning is necessary, the technique is similar except that the oral catheter is used.

The peroxide and water is used for oral irrigations. This is especially necessary where intraoral surgery has been performed. The soft rubber extension on the Asepto syringe prevents trauma to the intraoral tissues.

Fig. 3.—Apparatus arranged on tray, ready for use.

Fig. 4.—Tracheal toilet complete. Nurse is wiping secretions off the outside of the aspirating catheter with sterile sponge.

The saline in the fourth bottle serves two purposes: first, to rinse out the oral cavity after the peroxide irrigations and, second, to moisten the protective bib (Fig. 1) which the patient wears over the tracheotomy when he is ambulatory. Moistening the bib helps humidify the inspired air.

The inner tracheotomy tube may require frequent cleanings during the first few postoperative days. When this becomes necessary, the inner tube is removed and placed into a basin (emesis) containing peroxide and water. This helps soften the mucus within the lumen of the tube. The tube is then washed with green soap and water. A small, flexible brush is used for cleaning the inside of the tube. This is followed by scouring with a cleanser. The tube is thoroughly rinsed and allowed to boil in a sterilizer for two minutes. The inner tube is then returned to the patient as quickly as possible. This entire procedure should not take more than four or five minutes.

During the immediate postoperative period, Alevaire (an alkylaryl polyether alcohol detergent) is administered to the patient through a nebulizer. A plastic cone is attached to the long, flexible tube of the nebulizer. The cone is fastened loosely over the tracheotomy opening. The nebulus humidifies the inspired air, thins out mucus

Bacteriological Studies in Tracheotomy Care

Solutions Used for Rinsing and Storage of Tracheotomy Suction Catheters	Number of Patients Using Each Solution	Culture of Solutions After 6 Hours of Use		Culture of Solutions After 24 Hours of Use	
		Sterile	Growth of Organisms	Sterile	Growth of Organisms
Benzalkonium, 1:2000	5	5	0	5	0
Benzalkonium, 1:5000	7	7	0	7	0
Benzalkonium, 1:8000	7	7	0	7	0
Benzalkonium, 1:10,000	6	6	0	6	0
Benzalkonium, 1:20,000	7	5	2	4	3
Isotonic saline	6	0	6	0	6

1. All solutions were found to be sterile prior to use.
2. In most cases, the organism recovered in the contaminated solutions corresponded to one or more of the organisms recovered from the patient's tracheotomy.

secretions, and prevents tracheal incrustations.

To maintain a clean and fairly aseptic set-up, all equipment, i. e., the bottles, catheters, syringes, and solutions, is replaced each morning with a completely new sterile set-up.

Bacteriological studies were done to determine the proper dilution of aqueous benzalkonium necessary to maintain the sterility of the tracheal and oral aspirating catheters. Different dilutions of aqueous benzalkonium solutions were tested. The nurses and attendants administered tracheotomy care as outlined above. Samples of the solutions were taken from both the tracheal suction and oral suction bottles, 6 hours and 24 hours after the set-ups were changed, and these were cultured. Benzalkonium solutions of 1:2000, 1:5000, 1:8000, and 1:10,000 remained sterile even after 24 hours. A 1:20,000 solution of aqueous benzalkonium permitted growth in three of seven bottles tested. When only isotonic saline was used, all cultures showed growth in six hours (Table). It was concluded that a solution of 1:10,000 aqueous benzalkonium was adequate for maintaining sterility. To date, no untoward reactions or irritations of the tracheal mucous membrane have been noted.

In summary, the foregoing is an adequate and efficient method of tracheotomy care. The necessary equipment is simple and may be found on most hospital floors. Studies have shown that an aqueous benzalkonium dilution of 1:10,000 is adequate for soaking and rinsing aspirating catheters. This dilution not only maintains sterility but also appears to be nonirritating.

Roswell Park Memorial Institute.

Case Reports

Fever of Obscure Origin

Report of Case of Acute Mastoiditis Masked by Antibiotics

EUGENE Q. PARR, M.D., and O. ERIK HALLBERG, M.D., Rochester, Minn.

Since the advent of antibiotics and the sulfonamides, acute mastoiditis has become increasingly uncommon as a complication of acute otitis media. In this era with its abundance of easily administered, specific and broad-spectrum antibiotics, it is easy for the physician to use these when fever is the only clinical finding. Antibiotics, however, may mask almost completely the common manifestations of both acute and subacute mastoiditis. The purpose of this report is to present a case of this type. The diagnostic problem was fever of undetermined origin and the infectious disease obscured by antibiotics progressed to abscess formation. The problem of fever of obscure origin will become commoner unless the antibiotic drugs are used more discriminately and specifically and the changes in symptoms of impending serious illness from the classic ones are recognized promptly.

Report of Case

An 8-year-old girl was admitted to the Mayo Clinic on May 9, 1956, because of unexplained fever for six weeks, associated with anorexia, malaise, fatigue, and anemia. She had suffered from numerous episodes of bronchitis and sinusitis each year since she was 6 months of age. Two years before admission her family moved to a climate of lower humidity, and thereafter she experienced fewer attacks. She had suffered from mesenteric adenitis one and one-half years before admission, and tonsillectomy and adenoidectomy had been performed about six months later. Irrigations of the maxillary sinuses had been performed twice during the year before admission, with improvement of the sinusitis.

Early in March, 1956, the child became ill with the measles, and one week later, while she was recovering, right acute otitis media developed. She was given penicillin. The findings referable to acute otitis subsided in a few days, but for the next six weeks her temperature ranged from 101 to 103 F. The ear apparently never drained. She complained of fatigue, malaise, weakness, and anorexia and was confined to bed most of the time. She had received since the onset penicillin first and later erythromycin. Blood studies made by her physician indicated leukocytosis and mild anemia. She was referred to the clinic because of fever of undetermined origin.

The physical examination revealed a pale, well-nourished, well-developed child who appeared seriously ill. Her temperature was 100.5 F by mouth; blood pressure was 94 mm. of mercury systolic and 70 mm. diastolic, and her pulse rate was 86 beats per minute. An audiogram showed moderate deafness of conduction type on the right. The right tympanic membrane appeared intact, dull gray, thickened, and scarred, but there was no evidence of drainage or narrowing of the canal. She had pain in the right temporomandibular joint on extreme opening of the mouth, and pressure over this same joint also produced pain. The cervical nodes were enlarged bilaterally, but other nodes were normal. The heart, lungs, and abdomen appeared normal on examination.

Urinalysis disclosed nothing abnormal. The value for hemoglobin was 11.8 gm. per 100 cc. of whole blood, erythrocytes numbered 4,120,000 and the leukocytes 18,800 per cubic millimeter

Accepted for publication Sept. 19, 1956.

Fellow in Surgery, Mayo Foundation (Dr. Parr). Section of Otolaryngology and Rhinology, Mayo Clinic and Mayo Foundation (Dr. Hallberg). The Mayo Foundation, Rochester, Minn., is a part of the Graduate School of the University of Minnesota.

of blood. The differential blood cell count revealed 79% polymorphonuclear leukocytes, 15% lymphocytes, and 6% monocytes. The platelets numbered 279,000 per cubic millimeter, and the sedimentation rate of erythrocytes was 109 mm. in one hour by the Westergren method.

Roentgenologic examination showed nothing abnormal in the chest but indicated probable subacute mastoiditis. There was diffuse clouding with evidence of decalcification of the mastoid cell walls. The clinical impression was masked mastoiditis on the right; myringotomy followed by exploration of the right mastoid bone was advised.

On May 14, 1956, myringotomy disclosed that the right middle ear was completely dry. Through a postauricular incision, the mastoid cortex was opened and approximately 5 cc. of foul-smelling pus exuded under pressure. The entire mastoid bone was diseased. It was necessary to remove the tip and a long row of diseased cells from behind the sigmoid sinus. The sigmoid plate was removed and the sinus wall was revealed to be thickened and covered with granulation tissue. However, the wall was compressible and no thrombus seemed present. The mastoid antrum was completely blocked with swollen membrane and granulation tissue, which apparently prevented the pus from draining into the middle ear. The wound was closed with absorbable surgical (gut) and nonabsorbable surgical (silk) sutures. During the operative procedure, the patient was given 500 cc. of whole blood.

Micrococcus and Streptococcus pyogenes were cultured from the pus obtained at operation. Postoperatively, the patient was given 600,000 units of procaine penicillin G and 0.5 gm. of dihydrostreptomycin intramuscularly each day for one week. An audiogram on the fifth postoperative day showed no change in the air-conduction deafness. Her temperature became normal on the third postoperative day and remained so during the rest of the period of observation. She was dismissed from our care on May 19, 1956.

Comment

Williams,[1] in 1932, reviewed 585 cases of acute and subacute mastoiditis in which operation was performed at the Mayo Clinic. In 41 (7%) of these cases, disease was classified as masked mastoiditis. In addition, he reviewed the literature to determine the principal signs and symptoms of masked mastoiditis. He gave these as follows: (1) history of preceding infection of the upper respiratory tract or the middle ear; (2) weakness, anorexia, and loss of weight; (3) fever, intermittent or sustained; (4) lancinating pain referred to the occiput along the zygoma into the neck and into the temporal region; (5) sagging of the posterosuperior wall of the external auditory canal, and (6) a tendency to impairment of hearing. Our patient fulfilled these criteria with exception of items 4 and 5.

The history of a recent infection of the ear should always be sought, especially in children, when a physician is faced with the problem of fever of undetermined origin. A clue to the underlying problem may be given by the child's parent, who may have observed that her child does not hear as well as before the onset of illness. Fine[2] has suggested the use of a simple speech test to reveal unilateral loss of hearing. This test may be used for children who are more than 4 years of age and may be an important factor in discovering and treating early deafness in children. Our patient did not complain of loss of hearing, but it was obvious on speech testing that hearing was impaired in the right ear. Later, an audiogram confirmed that she had a 30-db. loss of hearing.

Aural discharge is an uncommon finding in masked mastoiditis because either the antibiotics have suppressed the bacterial growth in the middle ear or it has been blocked off by disease process in the mastoid antrum, as was evident in this case. If aural drainage occurs, it may be sterile and scant, and on discontinuing use of the antibiotic,[3] it may increase both in quantity and odor.

In our case, the tympanic membrane gave evidence of previous inflammation. It was dull gray, thickened, and scarred. When fluid is present in this type of ear for as long as several weeks, it is predisposed to chronic adhesive otitis media,[4] with subsequent loss of hearing of the air-conduction

type. When masked mastoiditis was seen prior to the use of antibiotics, deep mastoid bone ache and sagging of the posterosuperior canal wall were often the only clues to underlying disease. The latter finding indicated the additional involvement of the mastoid antrum.

Mastoiditis can occur without previous acute suppurative otitis media. Hallberg and Thornell[5] have reported such a case. Their explanation, which was first given by Hempstead,[6] was that the infection arises by direct extension from the nasopharynx to the Eustachian tube and middle ear, and through the mastoid antrum to the cells. In our case, the mastoid cavity was sealed off from the middle ear by swollen mucous membrane and granulation of the antrum. The effect of the antibiotics was apparently sufficient to suppress the infection sufficiently to prevent it from breaking into the middle ear and probably perforating the tympanic membrane.

"In most instances," according to Richardson,[7] "the radiologic haziness is revealed at operation as being granulations with thickened bony septa. All of this is on the surface of the normally delineating boundaries of the mastoid cavity." He also noted that the roentgenologic differentiation between the normal and the diseased mastoid was a particular problem in the young adults who had received treatment. He concluded, "a diseased condition existed if in the young adult there was seen thick, hazy cellular partitions, some arterial softening, increased density of the bone surrounding the posterior canal and if the comparative film was normally thin in trabeculae with changes equal in the tip views."

Most of the failures in the treatment of acute otitis media with subsequent development of mastoiditis[8] are due to inadequate, indiscriminate, and unwise administration of antibiotics. Inadequate treatment is considered to be administration of the agent in an insufficient daily quantity and duration. Indiscriminate use of antibiotics in illnesses which present fever as the only clinical finding may obscure the true nature of the disease, as seen in this case. There is also the problem of the organisms becoming resistant to the antibiotics.

Many patients who have acute mastoiditis are treated adequately with antibiotics, yet fail to respond, as indicated by persistent fever, pain, leukocytosis, and roentgenologic evidence of inflammation. The cause of failure in a limited number of cases may be due to resistant organisms or the fact that irreversible pathologic changes have already taken place in the mastoid.

Successful treatment of acute otitis media is dependent chiefly on the daily administration of an adequate quantity of antibiotics. The daily dose is based on both the severity of the infection and the recommended dosage per pound of body weight. In our experience, a patient with uncomplicated acute otitis media should receive antibiotics for several days after the infection has entirely subsided. Material draining from the external ear canal should be submitted for culture and antibiotic sensitivity tests. Then the most effective antibiotic may be selected. The patient must always be questioned about previous allergic reactions. Myringotomy is recommended in the presence of bulging tympanic membrane, and for it anesthesia should be administered to patients less than 16 years of age.

Summary

The case of an 8-year-old girl with acute mastoiditis masked by the administration of an antibiotic for six weeks is reported. The clinical picture included fever of undetermined origin for six weeks, generalized weakness, anorexia, and mild anemia. The diagnosis was based on a history of acute right otitis media six weeks previously, subsequent partial loss of hearing in the same ear, slight tenderness to pressure over the right temporomandibular joint, and roentgenologic evidence of subacute mastoiditis. Operation disclosed extensive involvement of the mastoid with pus under pressure. The significance of the principal

signs and symptoms of masked mastoiditis and the modes of onset are discussed together with the common causes of failure in the antibiotic treatment of acute otitis media and acute mastoiditis, and recommendations for adequate treatment.

Mayo Clinic.

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Abstracts from Current Literature

Ear

FORMATION, CIRCULATION AND CHEMICAL PROPERTIES OF THE LABYRINTHINE FLUIDS. L. CITRON, D. EXLEY, and C. S. HALLPIKE, Brit. M. Bull. 12:101 (May) 1956.

Volume 12, Number 2, of the *Bulletin* is devoted to the subject of neuro-otology. Since it is impossible to abstract every article therein, a few were chosen which may be of interest to the otologist, although men who work in neurology as well as in otology will find all the essays of interest. The eminent C. S. Hallpike is scientific editor of the series.

The biochemistry of the endolymph has to do with the high potassium and low sodium concentrations. These data seem unique for extracellular fluids and are closely related to like fluids in the body. ". . . strong support for the view that the endolymph is produced by the secretory activity of some specialized cells . . . in the stria vascularis (Guild) or in the saccus endolymphaticus cannot at present be stated. It is possible that both structures are concerned, and perhaps others too, not yet identified."

This is a study in pure science, is esoteric, and therefore will be stiff reading for most clinicians. The accompanying photomicrographs of the labyrinth are excellent. There are some 20 references to the literature.

I. W. VOORHEES, New York.

LOUDNESS RECRUITMENT. M. R. DIX, Brit. M. Bull. 12:119 (May) 1956.

The loudness recruitment phenomenon was first described by E. P. Fowler Sr., in 1928. It occurs in persons who suffer from some type of nerve deafness. Such subjects, even though they cannot understand speech at ordinary levels, complain of excessive loudness if these levels are exceeded. Recruitment is best studied in monaural deafness with binaural telephone receivers which can be shut off from either ear, with independent adjustment of the intensities. The later work of Dix, Hallpike, and Hood seems to show that there is involvement of the hair cells in the organ of Corti.

Many researchers have confirmed that the value of the recruitment concept is correct for deafness due to disease of the end-organs, and is absent in cochlear nerve fiber degeneration. There is consistent presence of loudness recruitment in conditions other than Ménière's disease with a clearly defined laboratory labyrinthine pathology. The conditions noted are acoustic trauma, hereditary perceptive deafness, meningitis, and syphilis. It is now possible to show the site and nature of the lesion responsible for deafness. For example, in virus affections of the eighth nerve, in disseminated sclerosis, acoustic trauma, and otitic labyrinthitis, a differential diagnosis is possible by application of the recruitment phenomenon.

I. W. VOORHEES, New York.

DISCUSSION ON THE MANAGEMENT OF DEAFNESS IN THE YOUNG CHILD. EDITH WHETNALL, Proc. Roy. Soc. Med. 49:455 (July) 1956.

The Section of Otology met on Dec. 2, 1955, under the presidency of E. G. Collins, F.R.C.S.Ed. The speaker has had much experience; she states that for 150 years otologists have asserted that the hearing of a deaf child can be trained (Ernauld, 1768, et al.). It is possible for a child with severe loss of hearing to acquire speech under the right conditions. As for "essential frequencies" being mandatory if a child is to be taught, graphs and charts are shown which seem to deny this dictum. The matter is one for home instruction by the parents rather than for institutions. "Early" is the decisive word. Talking into the child's ears should start during the first weeks and be continued without let-up. The first year is the most timely to acquire speech through hearing. Sounds must be repeated loud enough and often enough to reach the child's sensorium. Even beginning when the child is 2 years of age may be too late to secure the best results. The emphasis is on listening rather than watching. What happens in the ears is not nearly so important as what happens

in the brain, i. e., auditory discrimination. A child makes vowel sounds and hears them; his attendants make vowel sounds, and he hears them and tries to imitate them. It is all part of the general process of learning, e. g., learning to manipulate playthings, to eat, to walk. After 3 years of age the facility for learning to speak diminishes unless the primary stage is fully set up. By age 5, it is difficult to teach speech to a child through his hearing if there has been no previous training; and by 7 it is nearly impossible.

Learning speech through hearing calls for the following: 1. A hearing aid, small and easily manageable, which should continually be worn so that sounds may be heard at all times, not just occasionally. 2. The child should start to use an aid at 5 or 6 months; a wait up to 6 months may make a decided pejorative difference. The young child likes his aid and feels disturbed without it; the older child often rejects his aid and is disturbed by it. Lipreading is of benefit, but the tendency is to concentrate on watching rather than listening. In the child over 7 who is severely deaf, the aid is of very little value except as a means of improving the tone of the voice, unless the child can already discriminate between words. Considerable individual effort will be required. 3. Environment is a major factor in training. Individual rather than group teaching is the rule for speech and hearing given together. Unless one can get speech sounds to the child, deafness is likely to persist. However, it is of value to have the deaf child "mix" with hearing children. 4. Size of aid is paramount. The more it looks like a toy, the more interest the child has in it, but it must be worn continuously. There are no contraindications to this, and no evidence of any damage to hearing or general body economy has been seen. Getting a child to listen, to concentrate, is the first requisite; the residual hearing, if any, must be cultivated. He must know that sounds have meaning, and so the aid should be continuously worn for at least a year. A few odd half-hours will get the training nowhere. Tests of hearing must be stated as to whether these have been taken before or after auditory training. 5. Usable hearing can be determined only after giving the child an aid and talking to him for days or weeks. 6. Tests of intelligence are important, but difficult to make. An I. Q. of 75 may, with persistence, be raised to 110 after some years. Training of teachers of the deafened is a highly specialized kind of education. No child is able to help himself unaided; he must be dependent upon well qualified help.

Discussion by Mr. I. S. D. Thomson: In his first nine months of life, the normal child watches the faces of those around him, but later pays more attention to speech. Up to 18 months the profoundly deaf baby continues to study faces, but begins then to lose communication with his parents. Less severe deafness may not be suspected until speech defects become apparent. Under 9 months, reflex response to sounds gives evidence of hearing but "loud sounds are frequently ignored." Tests given behind the child will cause him to look around if he hears. Cooperation of all friends, neighbors, attendants, and parents is paramount. It is a tough job which can wear down everybody before results begin to appear. Here Mr. Thomson gives data concerning his technique of training.

Dr. D. C. Kendall continued the discussion: Team work is of the highest importance, and requires aid from the otologist, physicist, electrical engineer, educationist, and psychologist. In recent years, early diagnosis and early teaching have been stressed. The period before 12 months is paramount in working with any "residual hearing." The handicap of deafness strikes at the very roots of intellectual and social development; therefore speech must be developed by every possible means. The first essential is to devise test techniques consistent with the mental level of the child at hand. Word lists as now prepared are helpful. Dr. Kendall has set up five degrees of deafness: (1) average loss of 30 db. or below; (2) average loss of 30 to 60 db.; (3) average loss of 60 to 90 db.; (4) average loss of 90 db., indicating profound deafness; (5) no response, indicating total loss of hearing. This authority thinks that "the right place for the deafened or partially deafened child is at home with parents et al., and not in a residential school." Parents must be specially trained to cope with their problem.

Discussion by Dr. C. S. Hallpike: The central nervous system is basic in all training. The first three years of life are highly important; therefore the home is the essential field of instruction, and parental education is paramount.

J. W. Voorhees, New York.

Larynx

THE USE OF CONTINUOUS NEGATIVE PRESSURE AFTER LARYNGECTOMY AND RADICAL NECK DISSECTION. F. J. PUTNEY, Surg. Gynec. & Obst. 103:244 (Aug.) 1956.

The postoperative use of continuous negative pressure via catheters inserted into the neck wounds was employed in 11 patients who had undergone laryngectomy, 9 patients with radical neck dissection, and 16 patients with combined laryngectomy and neck dissection. The actual drainage after these procedures was measured and found to vary between 110 and 155 cc., depending upon the particular type of operation. In the 36 patients in whom this technique was used, pooling of secretion beneath the skin flaps with secondary infection and breakdown of the wound was prevented. The length of hospitalization and the development of pharyngeal fistulas were thereby decreased.

FRIEDBERG, Chicago.

Nose

A METHOD OF REPAIR FOR INTRANASAL ENCEPHALOCELE. F. A. FIGI, WINCHELL MCK. CRAIG, and M. E. KURTH, Am. J. Surg. 92:48 (July) 1956.

The authors present a new or unreported method for treatment of intranasal encephalocele, which is a congenital defect with protrusion of brain tissue and the covering membranes through an opening in the skull leading into the nasal cavity. This operation begins with an inverted U-shaped incision extending upward along the nasobuccal folds and across the root of the nose. The flap is reflected, and the underlying bulging bone is removed. The thin-walled meningeal sac is dissected free down to its neck, which leads through the defect in the floor of the anterior nasal fossa. In a case reported by the author the defect was at the base of the frontal and ethmoid bones, hardly more than 2 cm. in diameter. The neck of the sac was severed after being ligatured. A thin dermatome graft from the anterior abdominal wall was wrapped about a mold of sponge rubber with the raw surface outward. After dusting the incision with a sulfonamide-penicillin powder the skin-graft mold was inserted. The reflected nasal flap was then sutured back into its original position, with the long ends of the sutures tied over the gauze dressing to give firm pressure, and a roller head cover-all was added. After 19 days the stent was removed under intratracheal anesthesia, by opening the primary U-shaped incision. The point of special importance is the two-step operation: 1. Ligation and removal of the encephalocele, and lining the cavity with a skin graft. 2. Continuity was established between the nasal fossa and the skin-lined cavity that had contained the encephalocele, instead of the more extensive intracranial operation which is customarily done.

SELTZER, Philadelphia.

RECONSTRUCTION OF SUBTOTAL NASAL DEFECTS. G. B. O'CONNOR and M. W. MCGREGOR, Am. J. Surg. 92:57 (July) 1956.

The authors say that subtotal nasal defects may be congenital or due to infections, injury, and to new growths (both benign and malignant). The loss of tissue varies in different cases. The type and the location of the lesion are important and demand that replacing tissue be closely similar to that which is lost, particularly in color, the amount of hair present, and other such details. The type of graft must be considered; there are free grafts, as the split-skin type; full-thickness grafts, and composite grafts, which are made up of various proportions of skin, fat, and cartilage. Skin flaps, where possible, have the advantage of their own blood supply and match the wound area. Flaps derived from the forehead and about the nose and eyes are particularly suitable for this type of reconstruction. Eight photographs illustrate the paper.

SELTZER, Philadelphia.

ALLERGIC RHINITIS AND NASAL POLYPS. ROBERT A. CHAIT, New York J. Med. 56:2405 (Aug. 1) 1956.

This is the 16th article in a series dealing with modern allergy. Allergic rhinitis is a phrase which describes a condition that in most respects resembles a "cold." Seasonal allergy, commonly called hay fever, is periodic, but the perennial type, or "atopic perennial rhinitis," is probably the most distressing because of its persistence. A positive family

history is common. Sneezing, nasal obstruction, and profuse watery discharge are worse in the morning and toward evening. Nasal obstruction and polypi are concomitant, but not always associated. In the search for relief, the patient often employs the wrong means, e. g., vasoconstrictor drops, ice packs, hot pads, etc. Such measures may worsen the condition because of abuse of the mucous membrane. Nasal smears commonly show eosinophilia. Its presence and degree, upon repeated examinations, may give one some idea of the extent and progress of the malady. Nasal polypi are a complication of allergic rhinitis. After removal, stained sections commonly show eosinophilia. Polyps can develop in the absence of infection. "A surprisingly large number of polyps were reversible. . . . They would appear only during the hay fever season." Chait further states that upon removal of offending foods and inhalants a diminution in size or the total disappearance of polyps resulted.

ABSTRACTER'S COMMENT: The author does not believe in nasal surgery for allergic rhinitis. He seems surprised that one still removes polypi. He will have no cauterization or ionization. It is true that too much of any of these measures is to be condemned. But in moderation they bring great relief.

I. W. VOORHEES, New York.

THE COMMON COLD AND ALLERGIC RHINITIS. ABNER M. FUCHS, New York J. Med. 56: 2529 (Aug. 15) 1956.

The so-called "minor infections" often develop into major health problems, since they precede and may aggravate serious illnesses. We must now differentiate between bacterial and viral causes. The National Institute of Health, Bethesda, Md., is taking the lead in this work, especially in regard to the APC syndrome (adenoid-pharyngeal-conjunctival). Laboratory difficulties exist because there is no satisfactory experimental animal save man and the ape. The cold virus seems to be about 40 to 50 mμ in diameter. It produces little or no antibody response; therefore, immunity cannot be achieved, and "one cold on top of another" is a common experience. This agent may be found in the nose and throat of persons without symptoms; therefore the "carrier" is probably a chief means of reproducing colds in other persons. This is not true of nasal allergies—these cannot be transferred. An allergic hereditary background is possible, that is, a predisposition. Colds are carried home by children from school, who may unwittingly infect the whole family. No such occurrence is possible with allergy. Cold virus paves the way for secondary invaders—Staphylococcus Streptococcus, et al.—to begin their deadly work. We can do little in the fight against the virus, but much against the secondary invaders. Chemotherapy, the sulfonamides, penicillin, and other antibiotics are helpful, but do little or no good against viruses; in fact, they may worsen allergies. Allergies cause itching of the eyes, nose, and throat. The mucous membrane is bluish, puffy, and covered by watery secretion. Viral colds cause headache, nasal obstruction, and an outpouring of watery discharge, followed later by pus with possible invasion of the entire airway, top to bottom.

The respiratory specialist can do much to alleviate both of these types of disability by obtaining a correct history, testing the skin, and by trial-and-error, reproducing the symptoms when the patient is symptomless, and then is brought into contact with the actual cause.

ABSTRACTER'S COMMENT: We are just beginning to differentiate between bacterial and viral origins of disease. However, "virus" may be just another name for bacteria which are ultramicroscopic and have atypical characteristics. Patients are hopelessly confused and so often speak of "my sinuses" when, in fact, they do not have sinusitis as a manifestation of allergy, but a combined bacterial and viral infection. They may have one without the other or suffer from symbiosis. What we now must determine is which serious diseases, many of them chronic, begin with "a cold." It has not been disproved that poliomyelitis, multiple sclerosis, and perhaps Parkinson's disease may originate in the upper respiratory tract. In all of these, the slogan should be "Search for prodromal colds!" We know that the two great portals of entry are via the respiratory or the alimentary tract for a great host of systemic diseases, e. g., rheumatic fever and arthritis in its various forms. Here is a vast field for the young, enthusiastic researcher.

I. W. VOORHEES, New York.

THE CONTROL OF INFECTION IN ALLERGIC RHINITIS. MILTON S. DUNN, New York J. Med. 56:2692 (Sept. 1) 1956.

Dunn emphasizes the importance of the nasal smear in differentiating nasal infection from allergy, and as a guide to subsequent therapy. A control series of 25 patients was placed on antiallergic regimen and kept under observation for six months. Both eosinophils and leucocytes were present in large numbers, with the former predominating. As the allergy came more and more under control, eosinophils disappeared and leucocytes became more predominant. The conclusion was that infection was the causative factor which prevented the allergy from clearing under antiallergic care. Antibiotic application by nasal spray is now known to be useful; therefore the patients were told to spray the nose with Biomydrin four times a day. Results were diminished rhinorrhea, decreased nasal congestion, and change in the mucus from viscous yellow or green to clear. Patients stated that all previous symptoms lessened, especially itching and postnasal drip. No undesirable effects were noted. Skin excoriation disappeared in from 48 to 72 hours. This medication was continued for four weeks and was then stopped. In a week or two recurrence was evident, but was promptly relieved by a second course. The addition of hydrocortisone to the antibiotic solution did not alter the nasal smear findings. Biomydrin is said to contain a mucolytic agent which liquefies the secretions and permits antiseptic action upon the nasal mucous membrane. The article is substantiated by one table and 11 references to the literature.

I. W. VOORHEES, New York.

Miscellaneous

HEMANGIOMAS AND THEIR SURGICAL TREATMENT. J. T. MILLS, Am. J. Surg. 92:75 (July) 1956.

Various types of angiomas are the commonest congenital lesions seen by plastic surgeons. Hemangiomas may be present at birth or appear soon after. They may occur anywhere on the body, but are often in relation to the nose. Usually very small at first, they increase in size with the growth of the child. They are raised slightly above the skin surface, and when small are often bright red, forming what is known as strawberry nevi. Large growths may not be limited to the skin, but extend into the subcutaneous tissues to form the cavernous type, sometimes becoming greatly enlarged and forming destructive masses. The author emphasizes the desirability of having treatment given by group association of a plastic surgeon, dermatologist, and radiologist, since other treatment than surgery may be sufficient cosmetically. The treatment should always be conservative, and various operative methods are described. The paper is well illustrated by 10 photographs. There is no bibliography.

SELTZER, Philadelphia.

BILATERAL FACIAL AGENESIA (Teacher Collins Syndrome). C. C. SNYDER, Am. J. Surg. 92:81 (July) 1956.

The author describes bilateral facial agenesis as a rare congenital condition with an obscure cause, many theories of which are discussed. Characteristic signs include a tendency of the upper eyelids to droop at the outer edges. All parts of the head appear large because the malar bones are underdeveloped though the nasal cavity is not affected. The anomalies of the maxillary and malar bones depress the middle of the face, causing the nose to appear unduly prominent. The ears may also be affected. Effective treatment consists of a routine rhinoplasty, which will produce a suitable cosmetic result in each case. The article is illustrated with eight drawings and four photographs. Twenty-four references are given.

SELTZER, Philadelphia.

RECENT ADVANCES IN KNOWLEDGE OF RESPIRATORY VIRUSES. ALFRED L. FLORMAN, New York J. Med. 56:2383 (Aug. 1) 1956.

Florman is affiliated with the North Shore Hospital, Manhasset, N. Y., and the Mount Sinai Hospital. He limits his present studies to those diseases which arise in the upper respiratory tract (and the lower airways) and are amenable to laboratory investigation. He contrasts viral with bacterial origins. Measles is, in the beginning, "a cold" which is a cloak for our ignorance. It is self-limited and usually leaves a permanent immunity. It has not

been well studied heretofore because there is no really satisfactory laboratory animal for experimentation and also there are difficulties in growing and maintaining laboratory specimens. We now have, however, a complement-fixation test and a new source of virus which give promise of early diagnosis and treatment of this still serious disease. It is a true respiratory infection which cannot be differentiated from any other until Koplik spots and the rash appear. Nevertheless, a ray of hope is offered in the finding in stained smears of nasal mucus large multinucleated cells, basophilic and with well-preserved nuclei somewhat resembling fused normal epithelial respiratory cells. We may yet obtain a "vaccine" not incomparable to that of the Salk in poliomyelitis.

Influenza viruses have been well studied, and Types A, B, C, and D are now recognized. They have much in common and yet are not alike immunologically. Type C seems milder clinically, fails to agglutinate guinea pig cells, but hemaglutinates with chicken, human, and hamster cells in cold rather than in normal room temperatures. Variations occur in influenza viruses, especially in their antigenic constitution (Type A). These "may reflect a series of mutants in a partially immune population." These variations in antigenic properties have given direction to the hope of making a satisfactory influenza vaccine. Adjuvants can now be safely prepared for human use (Salk), making the outlook "for a successful influenza vaccine once again bright."

A group called APC viruses will interest the ENT men. It is derived from adenoid-pharyngeal-conjunctival sources. One sees patients with temperature spiked to 103 to 104 F with mild sore throat and a nonbacterial culture. The conjunctivitis is of a mild, follicular type, frequently mononuclear. The syndrome lasts only a few days, with headache, listlessness, drowsiness, and malaise to the end of the fever. These APC viruses were probably first described in Washington, D. C., in 1920, but were known in Australia (1943) and in Colorado (1951).

Psittacosis has been known as parrot fever for many years. It is a viral disease and may be serious in that it is often undiagnosed until suspected birds come into evidence. Fatal cases show a patchy bronchopneumonia. One should remember that this disease may be found in a chicken-raising area.

Herpangina is an acute febrile illness with vesicles or small ulcers on the anterior pillars, soft palate, tonsils, or pharynx. It is seen in children during summer months and may become epidemic.

ABSTRACTER'S COMMENT: This is the seventh in a series of articles on "Recent Advances in Medicine and Surgery" appearing in the *New York State Journal of Medicine*, under the supervision of Editor Robert Turell. Some 63 references are cited. For many years ENT specialists have been fully aware of the upper airways as a portal of entry in systemic diseases. Many patients have "colds" which are followed by irreversible chronic conditions. It may be that multiple sclerosis, which is gaining recognition as to its high incidence, is one of these(?). Poliomyelitis is a proved instance.

I. W. VOORHEES, New York.

THE PSYCHOLOGIC ASPECTS OF HEADACHE THERAPY. ARNOLD P. FRIEDMAN, New York J. Med. 56:2392 (Aug. 1) 1956.

An emotional conflict may be converted into a physical dysfunction, thus leading to a mistaken diagnosis of organic disease. Patients aware of their anxiety are often unaware of how it affects their health. Repressed emotions, such as anger, which "burns one up inside," may be expressed as headache. Specific syndromes are encountered, e. g., migraine. This is a form of headache which is paroxysmal, periodic, unilateral, and throbbing. A cause may be found in dilatation of cranial arteries and pressure on nerves and vessels passing through a foramen which has become too small. But this alone is not sufficient to cause headache. Tension headaches are usually bilateral and are associated with constant periodic emotional conflicts. Suppression of the latter may produce a kind of electrical (?) discharge, with changes in the muscles and blood vessels of the head and neck. Post-traumatic headache may or may not be accompanied by dizziness, difficulty in concentration, and nervousness. It occurred in 60% of those who had head injuries. In all cases of headache it is most difficult to determine whether the psychologic factors are primary or secondary, but psychotherapy in some form is indicated.

ABSTRACTER'S COMMENT: ENT men are often puzzled by patients who complain bitterly of headache. We hunt for an organic cause in the sinuses or for personal history that may

make us suspect tumor or meningeal hyperplasia. We probably often overlook the distinctly psychic factors, and do not know what to do about them. However, it is too easy to try to explain everything on psychogenic grounds. We must keep the organic and the psychic in some sort of balance.

I. W. VOORHEES, New York.

HAY FEVER. MURRAY M. ALBERT, New York J. Med. 56:2556 (Aug. 15) 1956.

Albert prefers the word "pollinosis" to hay fever. It is a seasonal disease, first described in 1819 by John Bostock, who called it "summer catarrh." The present author states that "Hay fever is a hereditary disease characterized by seasonal attacks of sneezing, rhinorrhea, nasal congestion, itching, and tearing of the eyes." He recognizes three types: (1) spring, caused by tree pollen; (2) summer, engendered by grass pollen; (3) autumn, associated with ragweed pollen, so widely distributed by winds. Asthma seems to be the worst complication. This is merely an extension of the irritating "dust" to the lower airways, with the physiopathology of the mucous membrane of the upper airways. Hyposensitization is the treatment of choice, given preseasonally or perennially as the case requires, with the specific, known pollen extract. "This form of treatment, when combined with the present adjuvants, such as the antihistamines or steroids, affords relief in over 90 percent of the cases." Palliative or symptomatic treatment, nose drops, cortisone, and corticotropin (ACTH) are much more helpful when used with specific treatment than when used alone.

ABSTRACTER'S COMMENT: In few of the many articles one reads these days anent allergies is any mention made of cauterization of the inferior turbinates. One article even condemns this procedure because it causes scarring and destruction of surface cells of the mucosa. This is largely a theoretical objection. Every ENT man can testify to the very great relief it brings. This far outweighs any disadvantage, and the patient will tell you so.

I. W. VOORHEES, New York.

THE SURGICAL TREATMENT OF ESOPHAGEAL STRICTURE. L. D. MACLEAN and O. H. WANGENSTEEN, Surg. Gynec. & Obst. 103:5 (July) 1956.

Attention is again directed toward the frequency with which the acid-peptic diathesis is responsible for esophagitis, esophageal ulcer, and spontaneous esophageal perforation. Nineteen patients with lower esophageal stricture were treated by surgical procedures designed to decrease acid-peptic irritation of the lower esophagus; in sixteen the operation consisted in subtotal gastrectomy and three were treated with hiatal hernia repair and pyloroplasty. With a single exception all patients experienced great relief of symptoms of their esophageal stricture and striking improvement appeared in the postoperative esophagograms. Benefits have been maintained over a prolonged period of postoperative observation.

It was of interest to the authors to observe that, whereas the presence of a congenitally short esophagus was diagnosed roentgenographically in several patients, in none of these patients was the condition observed at operation.

Esophageal dilatation was performed once or twice in the early convalescent period. The necessity of ensuring a functional lumen by dilatation directly following operation is emphasized. Other observers who failed to find subtotal gastrectomy successful in the treatment of lower esophageal stricture did not employ dilatation during the early recovery phase. Adequate gastric emptying must also be provided, lest aggravation of esophagitis occur.

Finally, the authors present some preliminary microscopic studies suggesting that the depth of esophageal lesion in acid-peptic esophageal stricture involves primarily the superficial layers of the esophageal wall (mucosa and submucosa) in contrast to the stricture which results from corrosive caustic agents, in which the entire esophageal wall becomes involved by fibrosis.

FRIEDBERG, Chicago.

PARAPHARYNGEAL NEURILEMMOMA. D. O. GORE, R. RANKOW, and J. M. HANFORD, Surg. Gynec. & Obst. 103:193 (Aug.) 1956.

Neurilemmomas are tumors of the neurilemma or nerve sheath of Schwann. Although commonest in the head and neck area, they are less frequently encountered in the parapharyngeal region. In this location they arise most often from the vagus or cervical sympathetic nerves. The differential diagnosis of a firm, painless mass in the upper lateral

neck with associated bulging of the posterior-lateral pharyngeal wall may be difficult but should include neurilemmoma if there is medial displacement of the internal carotid artery as well as dysphagia and hoarseness.

Surgical exploration, employing a lateral cervical approach and frozen tissue section, is recommended. Complete removal of the tumor is usually possible, but postoperative complications of vocal cord paralysis, Horner's syndrome, facial or hypoglossal nerve weakness may occur. Partial resection may be advisable in rare cases if irreparable damage to the vagus nerve is to be avoided.

The occurrence of von Recklinghausen's disease coincident with neurilemmoma was found to be only 1.5%. Four patients with parapharyngeal neurilemmoma are described in detail, bringing the total number of reported cases to 51.

FRIEDBERG, Chicago.

THE CHARACTERISTICS OF REGENERATION OF RESPIRATORY EPITHELIUM. N. O. CORRELL and E. J. BEATTIE, Surg. Gynec. & Obst. 103:209 (Aug.) 1956.

A brief study of the regenerative characteristics of tracheobronchial epithelium was undertaken by using resected tracheal grafts in mongrel dogs. After removal of a segment of trachea approximately 14 cartilaginous rings in length, the autogenous graft was turned end for end and anastomosed to the trachea at the points of excision. This reversal of a 2 to 3 in. segment was performed in 18 dogs and autopsies done after various intervals in order to study the rate of regeneration of the respiratory epithelium. It was found that ciliated columnar cells disappeared, leaving a layer of cuboidal cells which, in turn, gave rise to normal-appearing ciliated columnar epithelium in three to four weeks. The direction of beat of the regenerated cilia is the same as that of the original cilia of the grafted segment. The rate and manner of regeneration of columnar ciliated epithelium are similar to that described in the paranasal sinuses.

FRIEDBERG, Chicago.

News and Comment

ANNOUNCEMENTS

It has been the policy of the A. M. A. ARCHIVES OF OTOLARYNGOLOGY to publish original articles of interest to its readers. The editors of this journal are not necessarily in agreement with all conclusions reached by the authors of articles published. The editors invite readers to submit letters expressing and justifying differences of opinion with published conclusions. A selection of these letters will be published with the writer's name in the journal. All letters should be addressed to the Editor-in-Chief.

The Pietro Calicetti Award.—Competition is officially open for an International Award in memoriam of Pietro Calicetti, late Professor of the Clinic for Otorhinolaryngology of the University of Bologna.

The prize will be awarded to the physician who published within the last four years (Nov. 1, 1952-Oct. 31, 1956) the best scientific work of undisputed value and recognized originality in the specialty of otorhinolaryngology.

The prize consists of the following:

(*a*) A gold medal, offered by Mrs. Ines Misley, widow of Professor Calicetti
(*b*) The amount of L. 1,000,000 ($1,600)
(*c*) A diploma attesting the awarding of the prize

The professorial staff of the medical faculty of the University of Bologna will serve as a jury. If no publication is judged worth the prize, the Jury is free not to award it.

The following documents should be sent to the Secretary of the University before April 10, 1957:

(*a*) A notarized application giving academic degrees of the competitor
(*b*) Six copies of the scientific paper (five of which are intended for the Jury)

The Chief Administrator is Dr. Sebastiano Mazzaracchio. The President is Prof. Felice Battaglia.

Books

Bronchology. Two volumes. Second edition, completely revised and amplified. By Andre Soulas and Pierre Mounier-Kuhn. Price: paper, 14,000 francs; cloth, 15,550 francs. Pp. 1146, with 486 illustrations (67 full pages in color). Masson & Cie, 120 boulevard Saint-Germain, Paris, 6e, 1956.

This book, in two volumes, represents a complete detailed discussion of bronchology. It not only can serve as a textbook but also as an encyclopedia to the bronchoscopist and to the internist. As Dr. Chevalier Jackson stated in his preface, "The book is extremely clear, rich in illustrations, the quality of each chapter and the contributions about each pathologic condition are so numerous that it is impossible to pass in review on each subdivision."

The first chapter deals with the anatomy, and the succeeding chapters progress through physiology, topography, radiology and bronchography, routine methods of examination, and the normal findings. The succeeding chapters deal with various pathologic conditions, such as hemorrhages from tracheobronchial inflammations, tracheobronchitis, and bronchitis, and the various bronchopathies, such as bronchiectasis, in both adults and children.

The subject of tuberculosis receives extensive consideration. Here, detailed description is provided with numerous illustrations.

The second volume deals with the subject of new growths (both malignant and benign), foreign bodies, malformations, and anomalies.

Each section is amplified by numerous detailed photographs and many magnificent color plates which illustrate each pathologic condition. Microscopic sections are also presented in detailed color, which can, at a glance, clearly describe the subject under consideration.

There is an extensive bibliography at the end of each chapter. These two volumes should be considered a MUST for both the bronchoscopist and the internist.

Symptoms and Diagnosis of Diseases of the Nose, Throat and Ear. By Prof. Dr. M. Schwarz Tubingen. Price, DM 48. Pp. 351, with 88 illustrations (some in color). George Thieme Verlag, Herdweg 63, Stuttgart, 1956.

The author presents a discussion of the diseases of the ear, nose, and throat on a basis of both symptomatology and diagnosis. Each division is considered on the basis of symptoms, with a description of these symptoms and a discussion of the various pathologic conditions which may produce each particular symptom. There is a detailed evaluation of each pathologic condition and also a differential diagnosis. Therapy, both medical and surgical, has not been considered. The photographs, both in black and white, and in color, help considerably in clarifying the text.

The first chapter deals with anatomic abnormalities of the ear, and progresses from the external ear to the middle ear and, finally, to the inner ear, discussing every type of pathology that may involve this area. Tinnitus and deafness (including the various methods of audiology with their evaluation) are discussed in detail. Labyrinthitis is discussed, and all tests are evaluated for the various pathologic conditions.

The same format is followed for the diseases of the nose, including nasal abnormalities, fracture, epistaxis, hay fever, and orbital complications, the causes and sequelae of which are presented.

The next section deals with pathology about the mouth, the throat, the esophagus, and the larynx. There is a detailed section discussing impairment of speech, also headaches, pyogenic infections, focal infections, lymph node swellings, anosmia, and many other conditions, such as optic neuritis, paralysis of the abducens, the facial, and the trigeminal nerves, and, finally, the shoulder syndrome.

This book is an excellent epitome of the pathologic conditions of the nose, throat, and ear with all associated syndromes. It is clearly presented and is recommended as an excellent reference book.

Chronic Otitis in Infants (Report to the Congress of the French Society of Otolaryngology, 1955). By Jean Jacques Debain, Piere Coussieu, Jean Bourdial, and Yves Lallemant. Price, not given. Pp. 287, with 36 illustrations. Libraire Arnette, 1955.

The authors present an extensive monogram on chronic otitis media in infants. They discuss pathologic anatomy, the various classifications, the clinical examination, medical and surgical treatment, indications for operation, and the results of surgical treatment. This is an extensive, detailed description of this problem, and it merits a careful reading.

The Labyrinth. By Joseph J. Fischer. Price, $6.00. Pp. 206, with 30 illustrations. Grune & Stratton, Inc., 381 Fourth Ave., New York 16, 1956.

Dr. Fischer has concisely reviewed the voluminous literature on the labyrinth and has added to this his own experience and thinking to produce a valuable text to all those interested in the subject of the labyrinth.

Careful attention has been given to consider the subject from theoretical and hypothetical considerations, on the one hand, and more immediate clinical applications, on the other. Wherever possible, the discussion has been separated according to this pattern. In this way neither approach has been neglected, while at the same time cumbersome argument, which might detract from the clarity of the discussion, has been avoided.

Each of the currently described techniques of neuro-otologic evaluation is fully presented. Their relative value is discussed from both the view points of others and of Dr. Fischer. This provides an excellent critique of these methods.

The poignant discussion of neurologic concepts is an aid to the otologist seeking a more firm footing on these grounds. The neurologist will similarly profit from the clear presentation of the otologic aspects of the subject.

An excellent bibliography is provided, and in a stimulating manner the author has drawn attention to problems which remain to be solved.

The History of Otolaryngology in Berlin. By W. Kindler, B. Krebs, and G. Homm. Price, DM 16.50 ($3.95). Pp. 98 (paper), with 21 illustrations. Georg Thieme Verlag, Herdweg 63, (14a) Stuttgart, 1956.

This is a historical review of the growth of otolaryngology in Berlin. The first section deals with otology and traces the history of this specialty and the influence of such men as Kramer, Lucae, Trautmann, Passow, von Eicken, and their respective schools. The review includes the various associates that have served these professors and their contributions to otology. There is also a description of the origin and progress of the various clinics, their development, and their role in the development of this specialty. Furthermore, there is a description of the origin and growth of the Berlin Otologic Society.

The second section deals with laryngology and rhinology. The influence is related of such men as G. Muller, Lehrfeld, Garcia, Turck, Czermak, Lewin, Tobold, Waldenberg, and Frankel and their associates Rosenberg, E. V. Meyer, Finder, and Holscher. These were followed by A. Hartman, Claus, Killian, and Kirstein. Killian's associates were Albrecht, Wefngartner, Seiffert, and Lantenschlager. Further progress in this field, particularly in rhinoscopy and rhinoplasty, was enhanced by the work of Halle, Joseph, Gluck, and others. After Passow's death, von Eicken finally was able to consolidate otology with rhinolaryngology as a single specialty. Lastly, the authors describe the development of the voice and speech clinics in Berlin under Gutzmann and Gutzmann, and others.

This is an interesting historical review and contains many interesting episodes that developed throughout the course of this particular specialty.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society

(Société Canadienne d'Otolaryngologie)

President: Dr. G. M. T. Hazen, 208 Canada Building, Saskatoon, Sask.
Secretary: Dr. G. A. Henry, 328 Medical Arts Building, Toronto.
Place: Banff Springs Hotel. Time: June 17-19, 1957.

International Bronchoesophagological Society

President: Dr. Theodor Hünermann, Düsseldorf.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Sixth International Congress of Bronchoesophagology.
Place: Philadelphia. Time: May 12-13, 1957.

International Congress of Audiology

Place: Leyden, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

International Course in Paedo-Audiology

Place: Groningen, Netherlands.
General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. José Gros, Havana, Cuba.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Sixth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: Brazil. Time: 1958.

Sixth International Congress of Otolaryngology

President: Dr. Arthur W. Proetz, 3720 Washington Blvd., St. Louis.
General Secretary: Dr. Paul H. Holinger, 700 N. Michigan Ave., Chicago 11.
Place: Hotel Statler, Washington, D. C. Time: May 5-10, 1957.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse, N. Y.
Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Erling W. Hansen, 30 S. Ninth St., Minneapolis.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

American Board of Otolaryngology

President: Dr. Frederick T. Hill, Professional Building, Waterville, Maine.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

American Broncho-Esophagological Association

President: Dr. Clarence W. Engler, 2323 Prospect Ave., Cleveland.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958 (afternoons only).

American Laryngological Association

President: Dr. LeRoy A. Schall, 243 Charles St., Boston 14.
Secretary: Dr. Harry P. Schenck, 326 S. 19th St., Philadelphia 3.
Place: Statler Hotel, Washington, D. C. Time: May 3, 1957.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Percy E. Ireland, 100 College St., Toronto, Canada.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

SECTIONS:

Eastern.—Chairman: Dr. Joseph Sullivan, Toronto, Canada.
Southern.—Chairman: Dr. Mercer G. Lynch, New Orleans.
Middle.—Chairman: Dr. Paul M. Moore, Cleveland.
Western.—Chairman: Dr. Sigurd v. Christierson, San Francisco.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37.
Secretary-Treasurer: Dr. Dean M. Lierle, State University of Iowa, Iowa City.
Place: Statler Hotel, Washington, D. C. Time: May 4, 1957.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY, INC.

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AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

President: Dr. Dean McAllister Lierle, University Hospital, Iowa City.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2.
Place: Palmer House, Chicago.

SOCIETY OF MILITARY OTOLARYNGOLOGISTS

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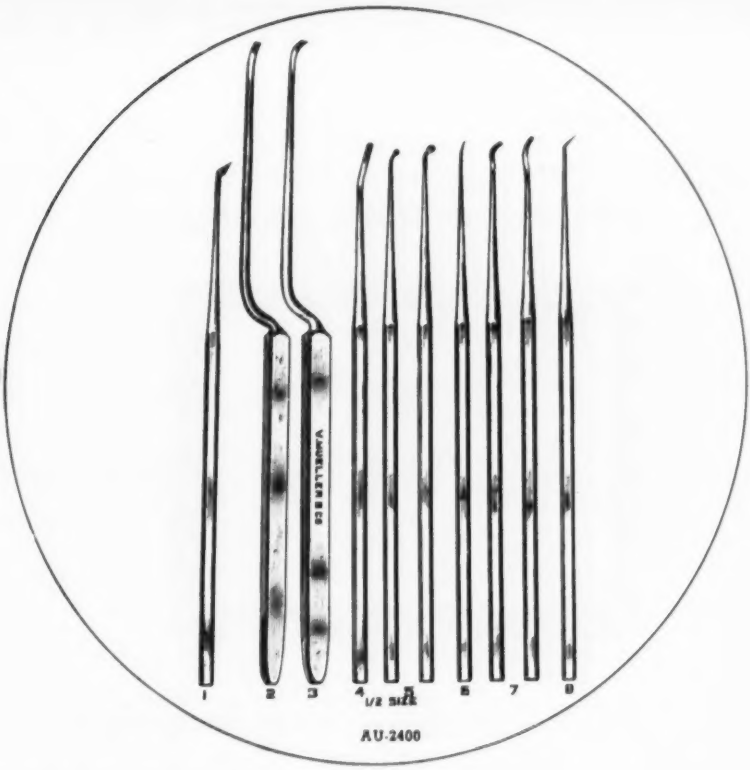
Secretary-Treasurer: Major Stanley H. Bear, M. C., 3810th USAF Hospital, Maxwell AFB, Ala.



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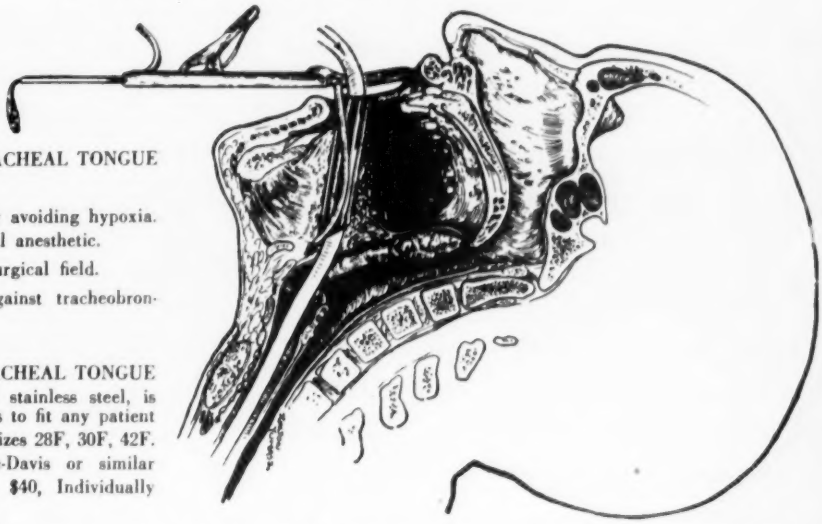

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*Barton, R. T.: Instrument for endotracheal anesthesia during oro-pharyngeal surgery. J. A. M. A. 153:1017 (Nov. 14) 1953.

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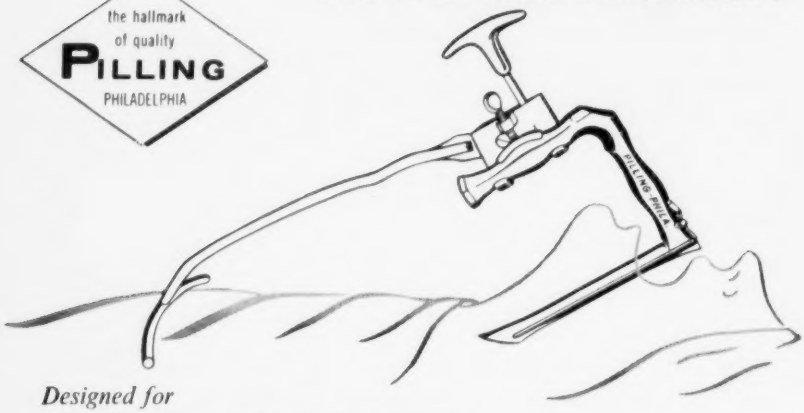

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Reference—R. B. Lewy: Acta Oto-Laryngologica, Vol. 46, Fasc. 1, 1956

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